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MEDICO-CHIRURGICAL

TRANSACTIONS.

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VOLUME THE FIFTY-NINTH.

MEDICO-CHIRURGICAL TRANSACTIONS.

ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

OFFICERS AND COUNCIL,
ELECTED MARCH 1, 1876.

President.
SIR JAMES PAGET, Bart., D.C.L., F.R.S.

VICE-PRESIDENTS.	EDWARD BALLARD, M.D. RICHARD PAYNE COTTON, M.D. CHARLES BROOKE, M.A., F.R.S. JOHN MARSHALL, F.R.S.
TREASURERS.	WILLIAM WEGG, M.D. JOHN BIRKETT.
SECRETARIES.	JOHN HARLEY, M.D., F.L.S. JOHN WHITAKER HULKE, F.R.S.
LIBRARIANS.	FRANCIS SIBSON, M.D., F.R.S. [DECEASED]. TIMOTHY HOLMES.
OTHER MEMBERS OF COUNCIL.	LIONEL SMITH BEALE, M.B., F.R.S. WILSON FOX, M.D., F.R.S. EDWARD HEADLAM GREENHOW, M.D., F.R.S. GRAILY HEWITT, M.D. WILLIAM OGLE, M.D. RICHARD BARWELL. JOHN COUPER. ARTHUR EDWARD DURHAM. GEORGE GASKOIN. THOMAS SMITH.

THE ABOVE FORM THE COUNCIL.

RESIDENT ASSISTANT-LIBRARIAN.
BENJAMIN ROBERT WHEATLEY.

A LIST OF THE PRESIDENTS OF THE SOCIETY
FROM ITS FORMATION.

FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS,

FOR THE SESSION OF 1876-77.

BARNES, ROBERT, M.D.
BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S.
CHOLMELEY, WILLIAM, M.D.
CHURCH, WILLIAM SELBY, M.D.
CLARK, FREDERICK LE GROS, F.R.S.
CURLING, THOMAS BLIZARD, F.R.S.
DAVIS, JOHN HALL, M.D.
FAGGE, CHARLES HILTON, M.D.
FERGUSSON, SIR WILLIAM, Bart., F.R.S.
FORSTER, J. COOPER.
GREENHALGH, ROBERT, M.D.
HABERSHON, SAMUEL OSBORNE, M.D.
HARLEY, GEORGE, M.D., F.R.S.
HAWKINS, CÆSAR HENRY, F.R.S.
HEWETT, PRESCOTT GARDNER, F.R.S.
HILTON, JOHN, F.R.S.
HOLTHOUSE, CARSTEN.
HUTCHINSON, JONATHAN.
PAVY, FREDERICK WILLIAM, M.D., F.R.S.
POLLOCK, JAMES E., M.D.
POWER, HENRY.
RADCLIFFE, CHARLES BLAND, M.D.
REES, GEORGE OWEN, M.D., F.R.S.
RINGER, SYDNEY, M.D.
SANDERSON, J. BURDON, M.D., F.R.S.
SAVORY, WILLIAM SCOVELL, F.R.S.
SIEVEKING, EDWARD HENRY, M.D.
SIMON, JOHN, D.C.L., F.R.S.
SMITH, SPENCER.
SOUTHEY, REGINALD, M.D.
THOMPSON, REGINALD, M.D.
THOMPSON, E. SYMES, M.D.
TILT, EDWARD JOHN, M.D.
WEST, CHARLES, M.D.
WILKS, SAMUEL, M.D., F.R.S.
WOOD, JOHN, F.R.S.

FELLOWS

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

P.—President.	V.P.—Vice-President.
T.—Treasurer.	S.—Secretary.
L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1876.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846   *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1851   *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847   ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1852 ADAMS, WILLIAM, Consulting Surgeon to the National
      Orthopædic Hospital, Great Portland street; 5, Henrietta
      street, Cavendish square. C. 1873-4. *Trans.* 2.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician
      to the Manchester Royal Infirmary; Cliff Point, Lower
      Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L.,
      late H.M.'s Envoy Extraordinary at the Court of Pekin.
      Trans. 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S.,
      Lecturer on the Practice of Physic at the Leeds School
      of Medicine, and Physician to the Leeds General Infir-
      mary; 35, Park square, Leeds. *Trans.* 3.

1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for
      Epilepsy and Paralysis; 18, Bryanston street, Portman
      square. *Trans.* 2.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill,
      Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on
      Medicine at, St. Bartholomew's Hospital; 22, Harley
      street, Cavendish square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1870 ARNOTT, HENRY, Chichester.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady
      Bank, Fifeshire, and 36, Sussex gardens, Hyde park.
      L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6.
      P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly. C.
      1821, 1830-31.

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hos-
      pital for Women; 1, Upper Wimpole street, Cavendish
      square.

Elected

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. *Trans.* 2.

1869 BAKEWELL, ROBERT HALL, M.D., Dunedin, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General, [6, Whitehall yard.] C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., *Vice-President*, Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.

1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.

1862 BARKER, EDGAR, 21, Hyde park street.

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to Charing Cross Hospital, and to the Hospital for Sick Children; 10, Montague street, Russell square.

Elected

1861   BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 31, Grosvenor street. *Trans.* 4.

1864   BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1840   BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859   BARWELL, RICHARD, Surgeon to the Charing Cross Hospital; 32, George street, Hanover square. C. 1876. *Trans.* 3.

1844   †BASHAM, WILLIAM RICHARD, M.D., Senior Physician to the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. T. 1871. *Trans.* 2.

1868   BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874   BAXTER, EVAN BUCHANAN, M.D., Assistant Physician to King's College Hospital; Professor of Materia Medica at King's College; 28, Weymouth street, Portland place.

1875   BEACH, FLETCHER, M.B., Medical Superintendent, Clapton Idiot Asylum, Lower Clapton.

1862   BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876. *Trans.* 1.

1860   *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.

1856   BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871   BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

Elected

1858   BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middle-
       sex County Lunatic Asylum, Hanwell; 26, Saint Peter's
       square, Hammersmith.

1819   †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne,
       Hants. C. 1832-3. V.P. 1854. *Trans*. 1.

1871   BELLAMY, EDWARD, Senior Assistant-Surgeon to, and Lec-
       turer on Anatomy at, Charing Cross Hospital; Professor
       of Anatomy in the Science and Art Department, South
       Kensington; 59, Margaret street, Cavendish square.

1847   BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and
       Mentone.

1845   †BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820   BERTIN, STEPHEN, Paris.

1872   BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Nor-
       folk and Norwich Hospital; 63, St. Giles's street, Nor-
       wich.

1865   *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool
       Royal Infirmary, and Lecturer on Clinical Surgery in
       the Liverpool Royal Infirmary School of Medicine; 2,
       Rodney street, Liverpool. *Trans*. 1.

1815   †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate
       of the University of London; 34, Park lane. C. 1825.
       V.P. 1828-9.

1854   BIRD, PETER HINCKES, F.L.S., Medical Officer of Health for
       the Fylde Union, West Lancashire; 4, Clifton terrace,
       Lytham, Lancashire, and 1, Norfolk square, Sussex
       gardens, Hyde park.

1856   BIRD, WILLIAM, Consulting Surgeon to the West London
       Hospital; Bute House, Hammersmith.

1849   BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to
       the City of London Hospital for Diseases of the Chest;
       48, Russell square. C. 1865-6.

1851   BIRKETT, JOHN, F.L.S., *Treasurer*, Inspector of Anatomy
       for the Provinces in England and Wales; 59, Green
       street, Grosvenor square. L. 1856-7. S. 1863-5.
       C. 1867-8. T. 1870-76. *Trans*. 8. *Sci. Com*.

Elected

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

1843 †BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.

1840 BLAKISTON, PEYTON, M.D., F.R.S.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.

1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow Gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.*

1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]

1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.

1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Oxford; 52, Bedford square.

Elected

1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 2.

1851 BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 56, Curzon street, Mayfair. *Trans.* 1.

1844 †BROOKE, CHARLES, M.A., F.R.S., *Vice-President*, Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72. V.P. 1875.

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., Laureate of the Academy of Sciences of Paris; New York, United States. *Sci. Com.*

1874 BRUCE, JOHN MITCHELL, M.D., Assistant Physician to the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 8, Old Cavendish street.

1867 BRUNJES, MARTIN, 42, Brook street, Grosvenor square.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica at the University of London; 23, Somerset street, Portman square.

1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Trans.* 8; *Pro.* 1. *Sci. Com.*

1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida Vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

Elected

1864   BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.

1864   BUCKLE, FLEETWOOD, M.D.

1839   BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.

1833   †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Trans.* 2.

1837   †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.

1873   BUTLIN, HENRY TRENTHAM, Surgical Registrar to St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 1.

1871   BUTT, WILLIAM F., 25, Park street, Park lane.

1818   BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.

1868   BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851   *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1861   CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to the Charter House; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.*

1874   CARR, WILLIAM, M.D., Lee Grove, Blackheath.

Elected

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; Surgeon to the Royal South London Ophthalmic Hospital; Professor of Pathology and Surgery at the Royal College of Sugeons; 69, Wimpole street, Cavendish square, W. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*

1868 CAVAFY, JOHN, M.D., Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; Physician to the Victoria Hospital for Children; 2, Upper Berkeley street, Portman square.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 58, Welbeck street, Cavendish square.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 24, Mount street, Grosvenor square. C. 1861. V.P. 1867. L. 1869-72. *Trans.* 1.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

Elected

1873 *CHISHOLM, EDWIN, Camden, near Sydney, New South
       . Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great
       Northern Hospital, and to the Margaret Street Infir-
       mary for Consumption; 63, Grosvenor street, Grosvenor
       square.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent,
       Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer
       on Clinical Medicine at, St. Bartholomew's Hospital;
       130, Harley street, Cavendish square.

1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on
       Medicine at, the London Hospital; 16, Cavendish
       square. C. 1875.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon
       to St. Thomas's Hospital; 14, St. Thomas's street,
       Southwark, and The Thorns, Sevenoaks. S. 1847-9.
       V.P. 1855-6. *Trans.* 5.

1848 CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C.
       1866.

1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., South-
       borough, Tunbridge Wells. *Trans.* 2.

1850 CLARKSON, JOSIAH, New Hall street, Birmingham.
       Trans. 1.

1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordi-
       nary to H.R.H. the Prince of Wales, and Surgeon-in-
       Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley
       street, Cavendish square. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish
       square. C. 1873.

1857 COATES, CHARLES, M.D., Physician to the Bath General and
       Royal United Hospitals; 10, Circus, Bath.

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free
       Hospital; 7, Suffolk place, Pall mall. *Trans.* 2.

Elected

1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.

COLLEY, DAVIES, see *Davies-Colley.*

1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.

1865 COOPER, ALFRED, Surgeon to the Royal Hospital for Diseases of the Chest, City road, Additional Surgeon for Out-patients to the Lock Hospital; Assistant-Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1819 COOPER, GEORGE, Brentford, Middlesex.

1873 COOPER, GEORGE HENRY CRESSWELL, F.R.C.S. Ed.; Surgeon to the Holloway and North Islington Dispensary; Surgeon-Accoucheur to the Royal Maternity Charity; 35, Compton terrace, Highbury.

1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.-P. 1873-4.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.

1853 CORY, WILLIAM GILLETT, M.D., Hengistbury House, Christchurch, Hampshire.

1847 †COTTON, RICHARD PAYNE, M.D., *Vice-President*, Consulting Physician to the Hospital for Consumption, Brompton; 33, Cavendish square. C. 1863. V.P. 1876.

1828 †COULSON, WILLIAM, F.L.S., Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

Elected

1864   COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860   †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876.

1862   COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.

1841   CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1868   CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.

1873   CREIGHTON, CHARLES, M.B., Anatomical Museum, Cambridge.

1869   *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874   CRIPPS, WILLIAM HARRISON, 53A, Pall Mall.

1847   CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Middlesex Hospital; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.

1868   CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square.

1862   CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.

1837   CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.

1860   CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.

1872   CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849   *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

Elected

1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Assistant Physician to King's College Hospital; Examiner in Anatomy at the University of London; 3, Warwick street, Cockspur street.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1852 CUTLER, THOMAS, M.D., Spa, Belgium.

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 1.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.

1846 DAVIES, FREDERICK, M.D., Upton House, Ryde, Isle of Wight. C. 1873.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.

1876 DAVIES-COLLEY, JOHN NEVILLE C., M.C., Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square.

1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; Examiner in Obstetric Medicine at the University of London; 24, Harley street, Cavendish square. C. 1869-70.

Elected

1867   DAY, WILLIAM HENRY, M.D., Physician to the Samaritan
       Free Hospital for Women and Children ; 10, Manchester
       square.

1846   *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859   †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and
       Lecturer on Clinical Medicine and Pathology at, St.
       George's Hospital; 11, Chesterfield street, Mayfair.
       C. 1874-5. *Trans.* 13. *Sci. Com.*

1839   †DIXON, JAMES, Consulting Surgeon to the Royal London
       Ophthalmic Hospital, Moorfields; Consulting Ophthal-
       mic Surgeon to the Asylum for Idiots ; Harrow Lands,
       Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4.
       C. 1866-7. *Trans.* 4.

1862   DOBELL, HORACE B., M.D., Consulting Physician to the
       Royal Hospital for Diseases of the Chest, City road ;
       84, Harley street. *Trans.* 2.

1845   DODD, JOHN.

1863   DOWN, JOHN LANGDON HAYDON, M.D., Physician to the
       London Hospital ; 39, Welbeck street, Cavendish
       square. *Trans.* 2.

1867   DRAGE, CHARLES, M.D., Hatfield, Herts.

1853   DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Ken-
       sington Mall.] *Trans.* 2.

1865   DRYSDALE, CHARLES ROBERT, M.D., Physician to the Far-
       ringdon Dispensary ; Assistant-Physician to the Metro-
       politan Free Hospital; 17, Woburn place, Russell
       square.

1865   DUCKWORTH, DYCE, M.D., Assistant-Physician to, and
       Lecturer on Skin Diseases at, St. Bartholomew's
       Hospital; 11, Grafton street, Bond street.

1845   DUFF, GEORGE, M.D., High street, Elgin.

1874   DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological
       Anatomy in King's College, London, and Physician to
       King's College Hospital ; 18, Devonshire street, Port-
       land place.

Elected

1874 DUKA, THEODORE, M.D., Surgeon-Major, H.M.'s Bengal Army; 38, Montagu square.

1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.

1871 *DUKES, CLEMENT, M.B. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales ; 62, Pall Mall.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital ; 82, Brook street, Grosvenor square. C. 1876. *Trans.* 5. *Sci. Com.*

1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital ; 38, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 DYER, HENRY SUMNER, M.D., Sennowe Hall, Guist, Norfolk. C. 1854-5.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B., Lond. Surgeon-Accoucheur to the Western General Dispensary; 5, Albion place, Hyde park square.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic ; 75, Harley street, Cavendish square.

1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle ; Coroner for Carlisle ; 35, Lowther street, Carlisle.

Elected

1848   ELLIS, GEORGE VINER, Professor of Anatomy in University
       College, London. C. 1863-4. *Trans.* 2.

1868   ELLIS, JAMES, M.D., Belle Grove Villa, Welling.

1854   *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal
       Household, Windsor; 14, High street, Windsor.

1842   †ERICHSEN, JOHN ERIC, F.R.S., late Professor of Clinical
       Surgery in University College, London, and Surgeon to
       University College Hospital; 6, Cavendish place, Caven-
       dish square. C. 1855-6. V.P. 1868. *Trans.* 2.

1874   EVANS, GEORGE HENRY, M.D., Assistant-Physician to the
       Middlesex Hospital; Assistant-Physician to the City
       of London Hospital for Diseases of the Chest, Victoria
       park; 29, Devonshire street, Portland place.

1845   EVANS, WILLIAM JULIAN, M.D., Pinner, Middlesex.

1875   *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick
       Children; 11, College square north, Belfast.

1864   FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and
       Lecturer on Pathology at, Guy's Hospital; 11, St.
       Thomas's street, Southwark. *Trans.* 6.

1869   FAIRBANK, FREDERICK ROYSTON, M.D., Hallgate, Don-
       caster.

1858   FALCONER, RANDLE WILBRAHAM, M.D., Physician to the
       Bath United Hospital; 22, Bennett street, Bath.

1862   FARQUHARSON, ROBERT, M.D., Lecturer on Materia Medica
       at St. Mary's Hospital; Physician to the Belgrave
       Hospital for Children; 23, Brook street, Grosvenor
       square.

1844   †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to
       H.M. the Queen; Physician-Accoucheur to H.R.H. the
       Princess of Wales; 12, Hertford street, Mayfair. C.
       1857. V.P. 1864. *Sci. Com.*

Elected

1872 FAYRER, SIR JOSEPH, K.S.I., M.D., F.R.S. Edin., Honorary
Physician to H.M. the Queen, and Physician to H.R.H.
the Duke of Edinburgh; Surgeon-Major, Bengal Army;
Examining Medical Officer to the Secretary of State for
India in Council; President of the Indian Medical
Board; 16, Granville place, Portman square.

1872 FENWICK, JOHN C. J., M.D.

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to, and
Lecturer on Medicine at, the London Hospital; 29,
Harley street, Cavendish square. *Trans.* 3.

1841 †FERGUSSON, SIR WILLIAM, Bart., F.R.S., Sergeant-Surgeon
to H.M. the Queen; Surgeon to King's College Hos-
pital; 16, George street, Hanover square. C. 1849-50.
V.P. 1863-4. *Trans.* 4.

1852 *FIELD, ALFRED GEORGE, Alverton Manor House, Stratford-
on-Avon.

1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint
Lecturer on Medicine at, the Westminster Hospital;
13, Belgrave road, Pimlico. C. 1871.

1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., Junior Physician
to the West London Hospital; 92, Wimpole street,
Cavendish square.

1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major. [6, White-
hall yard.]

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to
the Western General Dispensary; 30, Sussex gardens,
Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physi-
cian to the Birmingham General Hospital; 7, Waterloo
street, Birmingham. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Stafford-
shire Infirmary; Bedford House, Hanley, Staffordshire.

1848 †FORBES, JOHN GREGORY, 82, Oxford terrace, Hyde park.
C. 1868-9. *Trans.* 3.

Elected

1852 †FORSTER, JOHN COOPER, Surgeon to Guy's Hospital; Examiner in Surgery at the University of London ; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 FOX, EDWARD LONG, M.D., Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine ; Church House, Clifton, Gloucestershire.

1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen ; Physician in Ordinary to H.R.H. the Duke of Edinburgh ; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the University of London; 67, Grosvenor street. C. 1875-6. *Trans.* 3.

1871 FRANK, PHILIP, M.D., Cannes, France.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 29, Spring gardens.

1836 †FRENCH, JOHN GEORGE, '10, Cunningham place, Maida hill. C. 1852-3.

1876 FURNER, WILLOUGHBY, 111, King's road, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary ; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to Guy's Hospital; Assistant-Physician to the Hospital for Sick Children ; 14, St. Thomas's street, Southwark. *Trans.* 2.

1865 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital, 16, Connaught square, Hyde park. *Trans.* 2.

1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.

Elected

1867 GARLIKE, THOMAS W., Highfield, 126, Tulse hill, Brixton.

1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.

1851 GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6.

1819 GAULTER, HENRY.

1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874-5.

1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Trans.* 1.

1821 *GEORGE, RICHARD FRANCIS, 20, Marlborough buildings, Bath.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Physician to the Samaritan Free Hospital; 8, Upper Brook street, Grosvenor square.

1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 3.

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury Square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Heathfield, Ringwood, Hants. C. 1863.

Elected

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on
Pathology at, Charing Cross Hospital; Assistant-Phy-
sician to the Hospital for Consumption, Brompton;
74, Wimpole street, Cavendish square.

1875 GREENE, WILLIAM T., M.B., 218, Old Kent road.

1875 GREENFIELD, W. S., M.D., Demonstrator of, and Joint Lec-
turer on, Morbid Anatomy at St. Thomas's Hospital;
93, Wimpole street, Cavendish square.

1843 †GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and
Lecturer on Midwifery at, St. Bartholomew's Hospital;
Consulting Physician to the Samaritan Free Hospital
for Women and Children, and to the City of London
Lying-in Hospital; 72, Grosvenor street. C. 1871-2.
Trans. 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to,
and Lecturer on the Practice of Medicine at, the Mid-
dlesex Hospital; and Consulting Physician to the
Western General Dispensary; 14A, Manchester square.
C. 1876. *Trans.* 3.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Phy-
sician to the Westminster Hospital; Physician to the
In-Patients, Queen Charlotte's Lying-in-Hospital; As-
sistant-Physician to the Victoria Hospital for Children;
6, Curzon street, Mayfair.

1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque,
Paris.

1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S.,
Physician-Extraordinary to the Queen; Member of the
Senate of the University of London; Consulting Physi-
cian to Guy's Hospital; 74, Brook street, Grosvenor
square. C. 1864. V.P. 1874. *Trans.* 4.

1837 GULLY, JAMES MANBY, M.D.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and
Lecturer on Medicine at, Guy's Hospital; 70, Brook
street, Grosvenor square. S. 1867. C. 1869-70.
Trans. 3.

Elected

1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital, and to the Royal Westminster Ophthalmic Hospital; Standen House, Chute, Wilts. C. 1851. V.P. 1869.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals ; 13, Sussex place, Onslow gardens.

1836 HARDING, JOHN FOSSE, Ulverstone House, Uckfield, Sussex. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S. [25, Harley street, Cavendish square]. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.

1864 HARLEY, JOHN, M.D., F.L.S., *Secretary*, Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-6. *Trans.* 6.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine ; 38, Rodney street, Liverpool.

1841 †HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear and to the Freemasons' Female Charity; Aural Surgeon to the Great Northern Hospital; 3, George street, Hanover square. C. 1854.

1854 HAVILAND, ALFRED, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.

Elected

1870   HAWARD, J. WARRINGTON, Assistant-Surgeon to St. George's
       Hospital; Surgeon to the Hospital for Sick Children;
       5, Montagu street, Portman square. *Trans.* 1.

1828   †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to
       H.M. the Queen, and Consulting Surgeon to St.
       George's Hospital; 26, Grosvenor street. C. 1830-1,
       1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838   †HAWKINS, CHARLES, Inspector of Anatomical Schools in
       London; 27, Savile row, Burlington gardens. C.
       1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848   HAWKSLEY, THOMAS, M.D., Physician to the Margaret
       street Dispensary for Consumption and Diseases of the
       Chest; 6, Brook street, Grosvenor square.

1875   HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-
       Accoucheur and Assistant-Physician for Diseases of
       Women and Children to King's College Hospital; 17,
       Clarges street, Piccadilly.

1860   HAYWARD, HENRY HOWARD, Surgeon Dentist to, and
       Lecturer on Dental Surgery at, St. Mary's Hospital;
       38, Harley street, Cavendish square.

1861   HAYWARD, WILLIAM HENRY, Church House, Oldbury, Wor-
       cestershire.

1848   *HEALE, JAMES NEWTON, M.D., Medecroft, Winchester,
       Hants.

1865   HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery
       in University College, London; and Surgeon to Uni-
       versity College Hospital; 36, Cavendish square.

1850   HEATON, GEORGE, M.D., Boston, U.S.

1874   *HEATON, JOHN DEAKIN, M.D., Senior Physician to the
       Leeds General Infirmary, and Lecturer on Medicine at
       the Leeds School of Medicine; Claremont, Leeds.

1829   †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor
       square.

1821   HERBERSKI, VINCENT, M.D., Professor of Medicine in the
       University of Wilna.

Elected

1843   HEWETT, PRESCOTT GARDNER, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans. 7. Sci. Com.*

1855   HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876.

1872   HEYN, JULIUS CHARLES WILLIAM, M.D., 88, Lange Voorhout, the Hague, Holland.

1873   HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to Guy's Hospital; 38, Brook street, Grosvenor square.

1862   HILL, M. BERKELEY, M.B. Lond., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 55, Wimpole street, Cavendish square.

1867   HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1841   †HILTON, JOHN, F,R.S., Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans. 4.*

1859   HIRD, FRANCIS, Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

1861   *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1872   HOGG, FRANCIS ROBERTS, M.D., India.

1843   †HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874.

Elected

1868   HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-
        Physician to the Sussex County Hospital; 7, St. John's
        terrace, Brighton.

1861   HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road,
        South Hampstead.

1856   HOLMES, TIMOTHY, M.A. Camb., *Librarian*, Surgeon to, and
        Lecturer on Surgery at, St. George's Hospital; Surgeon
        in Chief to the Metropolitan Police Force; 18, Great
        Cumberland place, Hyde park. C. 1869-70. L.
        1873-6. *Trans.* 5. *Sci. Com.*

1846   HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lec-
        turer on Clinical Surgery at, the Westminster Hospital;
        Medical Officer of Health for Westminster; 14, Savile
        row, Burlington gardens. C. 1862-3.

1846   HOLTHOUSE, CARSTEN, 7, George street, Hanover square,
        and Balham hill house. C. 1863.

1865   HOWARD, BENJAMIN, M.D., Lecturer on Operative Surgery,
        and Surgeon to the Long Island College Hospital, New
        York; 134, West 34th street, New York.

1865   HOWARD, EDWARD, M.D.

1874   HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and
        Lecturer on Anatomy at, Guy's Hospital; Surgeon to
        the Evelina Hospital for Sick Children; 10, St. Thomas's
        street, Southwark. *Trans.* 1.

1857   HULKE, JOHN WHITAKER, F.R.S., *Secretary*, Surgeon to,
        and Lecturer on Surgery at, the Middlesex Hospital;
        Surgeon to the Royal London Ophthalmic Hospital,
        Moorfields; 10, Old Burlington street. C. 1871-2.
        S. 1876. *Trans.* 4. *Sci. Com.*

1857   HULME, EDWARD CHARLES, Woodbridge road, Guildford.
        Trans. 1.

1844   †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's
        wood. C. 1866-7.

1855   HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to
        Addenbrooke's Hospital; Professor of Human Anatomy
        and Physiology in the Cambridge University Medical
        School; Cambridge. *Trans.* 5.

Elected

1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.

1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. *Trans.* 6. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Athenæum Club, Pall Mall.

1876 IRVINE, JAMES PEARSON, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital; 3, Mansfield street, Cavendish square.

1871 JACKSON, J. HUGHLINGS, M.D., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital; 91, Harley street, Cavendish square.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

Elected

1841   JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825   JAMES, JOHN B., M.D.

1839   JEFFREYS, JULIUS, F.R.S., 9, Park villas west, Queen's road, Richmond, Surrey.

1840   *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851   JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Trans.* 3.

1851   JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."

1847   JOHNSON, GEORGE, M.D., F.R.S., Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-4. V.P. 1870. *Trans.* 10.

1868   JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.

1848   JOHNSTONE [JOHNSON], ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.

1862   JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1876   JONES, LESLIE, M.D., 3, Brighton Parade, Blackpool, Lancashire.

1875   *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University. [Agents: Messrs. D. Jones & Co., 2; Gresham buildings; Basinghall street.

1837   †JONES, THOMAS WILLIAM, M.D., 55, St. John's park; Upper Holloway. C. 1858.

Elected

1859   JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865   JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

1816   *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1872   KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex.

1848   *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1847   KEYSER, ALFRED, King's Hill, Berkhampstead.

1857   KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.

1851   KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*

1876   *KOCH, EDWIN LAWSON, M.D., Principal, Medical School of Ceylon; Colombo, Ceylon. [Agents: Messrs. Henry S. King & Co., 65, Cornhill.]

1855   LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.

1840   †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.

1865   LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square.

1873   *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

Elected

1841   *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862   LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816   LAWRENCE, G. E.

1843   *LEACH, JESSE, Moss Hall, Heywood, Lancashire.

1868   LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.

1843   †LEE, HENRY, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V. P. 1868-9. *Trans.* 13. *Pro.* 2. *Sci. Com.*

1822   †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Academy of Medicine, Paris; 15, The Avenue, Berrylands, Surbiton, and 28, Maddox street, Bond street. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1869   LEGG, JOHN WICKHAM, M.D., Physician to Casualty Department and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; 47, Green street, Park lane. *Trans.* 2.

1836   LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

1872   LIEBREICH, RICHARD, Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at St. Thomas's Hospital; 16, Albemarle street, Piccadilly.

1806   LIND, JOHN, M.D.

1872   *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871   LITTLE, LOUIS STROMEYER, Shanghai, China.

1870   LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819   LLOYD, ROBERT, M.D.

Elected

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., 4, Graham street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.

1867 MAC CORMAC, WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.

1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1866 MACGOWAN, ALEXANDER THORBURN, Kingswood park, near Bristol.

1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

Elected   .

1873 MacKellar, Alexander Oberlin, M.S.I., Assistant Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.

1854 *Mackinder, Draper, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1860 Maclean, John, M.D., 24, Portman street, Portman square.

1849 Maclure, Duncan Maclachlan, M.B., Lecturer on Physiology at the Westminster Hospital; Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.

1876 Macnamara, Charles, Surgeon to the Westminster Hospital; Surgeon Major Bengal Medical Service; late Examiner in Surgery at the Calcutta University; 13, Grosvenor street.

1842 Macnaught, John, M.D., 74, Huskisson street, Liverpool.

1876 Mallam, Benjamin, Percy Villa, 316, Camden road.

1855 Marcet, William, M.D., F.R.S.; Villa Bianca, Cannes. C. 1871. *Trans. 3. Sci. Com.*   .

1867 Marsh, F. Howard, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Bruton street, Berkeley square. *Trans. 2.*

1838 Marsh, Thomas Parr, M.D.

1851 Marshall, John, F.R.S., *Vice-President*, Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens, C. 1866. V.P. 1875-76. *Trans. 2.*

1864 Mason, Francis, Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans. 1.*

1869 Mayo, Charles, M.B., Colonial Surgeon at the Fiji Islands. [New University Club, St. James's street.]

1839 Meade, Richard Henry, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans. 1.*

1870 Meadows, Alfred, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square.

Elected

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent.

1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868-9. *Trans.* 2.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1873 MILNER, EDWARD, Surgical Registrar, St. Bartholomew's Hospital; 32, New Cavendish street, Portland place.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.

1844 †MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

1836 MOORE, GEORGE, M.D., Hastings.

1873 MOORE, NORMAN, M.D., Warden of the College and Lecturer on Comparative Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.

1874 MORRIS, HENRY, M.A. Lond., Senior Assistant-Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital ; 2, Mansfield street, Portland place. *Trans.* 1.

Elected

1851   MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General
         of Hospitals; Medical Inspector to the Local Govern-
         ment Board; and Member of the Senate of the University
         of Calcutta; 12, Durham villas, Kensington.

1868   MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer
         on Clinical Medicine at, Guy's Hospital; 6, Finsbury
         Circus. *Trans.* 1.

1856   MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., Phy-
         sician to, and Lecturer on Medicine at, St. Thomas's
         Hospital, Consulting Physician to the London Fever
         Hospital; Examiner in Medicine at the University of
         London; 79, Wimpole street, Cavendish square. C.
         1870-71. *Trans.* 3.

1875   MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 9, London
         street, Norfolk square, Hyde park.

1873   MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntress Row, Scar-
         borough.

1863   MYERS, ARTHUR B. R., Surgeon to the 1st Battalion Cold-
         stream Guards; Hospital, Vincent square, Westminster.

1876   NAPIER, WILLIAM DONALD, 22, George street, Hanover
         square, W.

1870   NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medi-
         cine in the University of Melbourne; 166, Collins
         street east, Melbourne, Victoria.

1835   †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace,
         York gate, Regent's park.

1843   †NEWTON, EDWARD [4, Upper Wimpole street]. C. 1863-4.

1868   NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

1849   NORMAN, HENRY BURFORD, Portland Lodge, Southsea,
         Hants.

1847   *NOURSE, WILLIAM EDWARD CHARLES, Surgeon to St.
         Mary's Hospital, Brighton; 11, Marlborough place,
         Brighton.

1849   NOVERRE, ARTHUR, 16, Park street, Grosvenor square.
         C. 1870-71.

Elected

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 30, Cavendish square. C. 1873. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, Derby.

1860 OGLE, WILLIAM, M.D., Medical Officer of Health for East Hertfordshire; 10, Gordon street, Gordon square. S. 1868-70. C. 1876. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.B., Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 2.

1875 OSBORN, SAMUEL, ; 17, Gresham park, Brixton.

1874 PAGE, HERBERT WILLIAM, M.B., M.C., Assistant Surgeon to St. Mary's Hospital; 28, New Cavendish street.

1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., *President*, Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. P. 1875-76. *Trans.* 9. *Sci. Com.*

Elected

1858   *PALEY, WILLIAM, M.D., Physician to the Ripón Dispen-
       sary; Ripon, Yorkshire.

1847   PARKER, NICHOLAS, M.D., Paris.

1873   PARKER, ROBERT WILLIAM, 8, Old Cavendish-street.

1841   PARKIN, JOHN, M.D. Rome; Temple Club, Arundel street.

1865   PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to,
       and Lecturer on Physiology at, Guy's Hospital; 35,
       Grosvenor street.

1869   PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and
       Lecturer on General Pathology at, St. Thomas's Hos-
       pital; 6, Savile row, Burlington gardens.

1845   †PEACOCK, THOMAS BEVILL, M.D., Physician to St. Thomas's
       Hospital; Consulting Physician to the City of London
       Hospital for Diseases of the Chest, Victoria Park; 20,
       Finsbury circus.   S. 1855-6.   V.P. 1867.   C. 1869,
       Trans. 2.

1856   PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830   PELECHIN, CHARLES P., M.D., St. Petersburg.

1855   *PEMBERTON, OLIVER, Surgeon to the Birmingham General
       Hospital, and Professor of Surgery at the Queen's
       College, Birmingham; 18, Temple row, Birmingham.
       Trans. 1.

1874   PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's,
       Sussex.

1870   PERRIN, JOHN BESWICK, Medical Tutor and Demonstrator
       of Practical and Surgical Anatomy, Owens College;
       51, Nelson street, Manchester.

1852   PHILLIPS, RICHARD, 27, Leinster square, Bayswater.

1846   PHILP, FRANCIS RICHARD, M.D. [Colby House, Ken-
       sington.]

1867   PICK, THOMAS PICKERING, Assistant-Surgeon to, and
       Lecturer on Anatomy at, St. George's Hospital; 7,
       South Eaton place, Eaton square.   *Sci. Com.*

1841   †PITMAN, HENRY ALFRED, M.D., Consulting Physician to
       St. George's Hospital, and to the Royal General Dis-
       pensary, St. Pancras; 28, Gordon square.   L. 1851-3.
       C. 1861-2.   T. 1863-8.   V.P. 1870-1.

Elected

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.D., Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street.

1843 POPE, CHARLES, M.D., The Rectory, East Harptree, Bristol.

1846 POTTER, JEPHSON, M.D., F.L.S., 6, Soho street, Liverpool.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Senior Assistant-Physician to, and Lecturer on Materia Medica at, Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 15, Henrietta street, Cavendish square. *Trans.* 1.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Sci. Com.*

1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 1, Pembridge place, Bayswater.

1874 PURVES, WILLIAM LAIDLAW, M.D., Aural Surgeon to Guy's Hospital; 7, Hanover street, Hanover square.

Elected

1850 QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton ; Member of the Senate of the University of London ; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen ; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital ; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital ; Physician to the National Hospital for the Paralysed and Epileptic ; 25, Cavendish square. C. 1867-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich ; 26, Queen Anne street, Cavendish square.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital ; the Pavement, Nottingham.

1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Petersham terrace], Queen's gate, South Kensington.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital ; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.

1869 REEVES, WILLIAM, 5, the Crescent, Carlisle.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital ; 38, Grosvenor street. C. 1870.

Elected

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B.

1869 RICKARDS, WALTER, M.D., Physician to the Royal Free Hospital; 8, Cavendish place, Cavendish square.

1845 †RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. *Trans.* 4.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1852 ROBERTS, JOHN, M.R.C.P., the Park, Westow hill, Upper Norwood.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

1843 RODEN, WILLIAM M.D., Morningside, Kidderminster, Worcestershire.

1850 ROPER, GEORGE, M.D., Physician to the Royal Maternity Charity; 6, West street, Finsbury circus.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

Elected

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, M.D., F.R.S., Knoll Hill, Prestbury, near Cheltenham.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 91, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh.

1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City.   C. 1871.   *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square.   C. 1872-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Jodrell Professor of Human Physiology and Histology at University College, London; 49, Queen Anne street, Cavendish square.   C. 1869-70.   *Trans.* 2.   *Sci. Com.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Cheltenham.

1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; 29, Duncan terrace, Islington.   *Trans.* 1.

1845 †SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square.   C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square.   C. 1871-2.   *Trans.* 4.   *Sci. Com.* 3.

Elected

1873 SCOTT, J. M. JOHNSTON, M.D., 14, College square, east, Belfast.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. *Trans.* 2.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London; 50, Torrington square. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *SHEARMAN, EDWARD JAMES, M.D., F.R.S. Edin., F.L.S., Consulting Physician to the Rotherham Hospital; Moorgate, Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, 4, Savile row. *Trans.* 4. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Trans.* 2. *Sci. Com.*

1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.

1842 †SIMON, JOHN, K.C.B., D.C.L., F.R.S., Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.

Elected

1865   SIMS, J. MARION, M.D., Surgeon to the New York State
       Women's Hospital; 267, Madison Avenue, New York.

1857   SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.

1872 · SMITH, GILBART, M.A., M.B., Physician to the Royal Hos-
       pital for Diseases of the Chest, City road; Visiting
       Physician to the Margaret Street Infirmary for Con-
       sumption; 68, Harley street, Cavendish square.

1866   SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hos-
       pital for Women; Physician to the British Lying-in
       Hospital; 2, Portugal street, Grosvenor square.

1835   SMITH, JOHN GREGORY, 23, Gloucester place, Greenwich.

1838   †SMITH, SPENCER, Surgeon to, and Lecturer on Clinical
       Surgery at, St. Mary's Hospital; 9, Queen Anne street,
       Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60.
       T. 1865.

1863   SMITH, THOMAS, Surgeon to, and Lecturer on Clinical
       Surgery at, St. Bartholomew's Hospital; Surgeon
       to the Hospital for Sick Children; 5, Stratford place,
       Oxford street, S. 1870-2. C. 1875-76. *Trans.* 3.
       Sci. Com.

1864   *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray,
       Kent.

1845   SMITH, WILLIAM, 70, Pembroke road, Clifton, Bristol.
       Trans. 1.

1847   SMITH, WILLIAM J., M.D., Consulting Physician to the
       Weymouth Infirmary; Greenhill, Weymouth, Dorset-
       shire.

1873   SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital,
       Greenwich.

1874   *SMITH, WILLIAM ROBERT [Royal County Hospital, Win-
       chester], 13, Crescent road, Plumstead, Kent.

1868   SOLLY, SAMUEL EDWIN [Ivy House, East Sheen].

·1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on
       Forensic Medicine at, St. Bartholomew's Hospital; 6, ·
       Harley street, Cavendish square.

Elected

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1874 SPARKS, EDWARD ISAAC, M.B. [Abroad.] *Trans.* 1.

1851 SPITTA, ROBERT JOHN, M.D. Lond., Clapham Common, Surrey. *Trans.* 1.

1875 SPITTA, EDMUND J., late Demonstrator of Anatomy at St. George's Hospital; Clapham Common.

1843 *SPRANGER, STEPHEN, Cape Town, South Africa.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board. [Greenford House, Sutton, Surrey.]

1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, WILLIAM, Jun., M.D., Professor of Surgery, Royal College of Surgeons, Ireland, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin, *Trans.* 1.

1843 STORKS, ROBERT REEVE, 'Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5.

1876 STRETTON, WILLIAM HARRIS, M.D., Physician to the Farringdon Dispensary; 8, Suffolk place, Pall Mall East.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square.

1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

Elected

1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark.

1852 TAYLOR, ROBERT, Surgeon to the Cripples' Home, Marylebone road; 7, Lower Seymour street, Portman square.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 2.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 3, Upper George street, Bryanston square. S. 1871-4. *Trans.* 1. *Sci. Com.*

1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

Elected

1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 4.

1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 8, Cranley place, Onslow square. *Trans.* 1. *Sci. Com.*

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 83, Park street, Grosvenor square.

1875 TIBBITS, HERBERT, M.R.C.P. Ed., Medical Superintendent of the National Hospital for the Paralysed and Epileptic; 26, Cavendish square.

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square.

1872 TOMES, CHARLES S., B.A., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.

1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Assistant-Physician to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., 3, Warrior square, St. Leonard's-on-Sea.

1870 VENNING, EDGCOMBE, Surgeon, 1st Life Guards; Knightsbridge Barracks, and 87, Sloane street.

Elected

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 43, Weymouth street, Porland place.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.

1867 *WALLIS, GEORGE, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road; 27, Weymouth street, Portland place.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

1851 WALTON, HAYNES, Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

Elected

1866 WARING, EDWARD JOHN, M.D., 49, Clifton gardens, Maida vale.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square. *Trans.* 1.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-6.

1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualeguay-chu, Entre Rios, Argentine Confederation.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 9. *Pro.* 1.

Elected

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Assistant-Physician to, and Lecturer on Botany at, St. George's Hospital; 37, Green street, Grosvenor square.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 †WILDBORE, FREDERIC, 245, Hackney road.

1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.

1863 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C.1849-50. V.P. 1860-1. P. 1873-4. *Trans.* 1. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Trans.* 3.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square.

Elected

1859 WILLIAMS, JOSEPH, M.D. Holmhurst, Cambridge park, Twickenham.

1868 WILLIAMS, WILLIAM RHYS, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Bethlehem Royal Hospital, Lambeth road.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxford-shire.

1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill, . Upper Norwood, Surrey.

1841 WOOD, GEORGE LEIGHTON, 28, Green park, Bath.

1851 WOOD, JOHN, F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; Examiner in Anatomy and Physiology at the University of Cambridge; 68, Wimpole street. C. 1867-8. *Trans.* 3.

1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8.

1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, 62, Bedford gardens, Kensington.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D., Professor of Materia Medica in the University of Edinburgh; Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.

1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.

1868 HOOKER, JOSEPH DALTON, M.D., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; President of the Royal Society; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

Elected

1847 OWEN, RICHARD, C.B., D.C.L., LL.D., F.R.S., Superinten-
dent of the Natural History Departments in the British
Museum ; Foreign Associate of the Academy of
Sciences of the Institute of France; Sheen Lodge,
Mortlake.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian
Professor of Mathematics in the University of Cam-
bridge; Secretary to the Royal Society, &c.; Lensfield
Cottage, Cambridge.

1875 STOKES, WILLIAM, M.D., D.C.L., LL.D., F.R.S., Regius
Professor of Physic at Dublin University; 5, Merrion
square north, Dublin.

1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural
Philosophy in the Royal Institution; Corresponding
Member of the Academies and Societies of Sciences of
Göttingen, Haarlem, Geneva, &c. ; Royal Institution,
Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1872 BERNARD, CLAUDE, Member of the Institute of France, and of the Academy of Medicine ; Professor of Medicine at the College of France ; Professor of General Physiology at the Museum of Natural History ; Rue de Luxembourg, 24, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1875 DRAPER, JOHN WILLIAM, M.A., LL.D., Emeritus Professor of Chemistry and Physiology in the University of New York ; 13, University Buildings, Washington square, New York.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden ; President of the College of Health, and Director-General of Hospitals ; Stockholm.

1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 HELMHOLTZ, H., Professor of Physics and Physiological Optics ; Berlin.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

Elected

1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1850 ROKITANSKY, CARL FREIHERR VON, M.D., Curator of the Imperial Pathological Museum, and Professor of Pathological Anatomy at the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

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ADVERTISEMENT.

REGULATIONS relative to the publication of the ' Proceedings of the Society.'

That, as a general rule, the Proceedings will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the Proceedings will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That ' The Proceedings of the Society ' may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the Proceedings. Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the Proceedings *only*, or in the Proceedings and Transactions; and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ON THE PATHOLOGY OF

CHOREA.

By W. HOWSHIP DICKINSON, M.D. Cantab., F.R.C.P.,

PHYSICIAN TO ST. GEORGE'S HOSPITAL AND TO THE HOSPITAL FOR
SICK CHILDREN; CORRESPONDING MEMBER OF THE
ACADEMY OF MEDICINE OF NEW YORK.

In the present nascent state of the pathology of the nervous system what is most wanted is the accumulation of observations ; general laws may hereafter become manifest and lines of classification show themselves; but as yet it is apparent that, putting aside the coarser injuries, our knowledge on this question is so far incomplete that it is hardly possible to trace, without intervals of conjecture, the progress of any disease belonging to this portion of the frame, from its origin in external or inherent influences to its close in fatal damage to necessary structures.

As a small contribution to a large want I propose to take the subject of Chorea, one which opportunity has brought in my way, and to describe the state of the nervous centres in a short series of cases, with such brief reference to other circumstances as may help to throw light upon the pathology of the disorder.

The instances are related as nearly as could be judged in the order of their acuteness, beginning with those in which death ensued at the earliest stages of the disease.

Case 1.—On the 12th of October, 1875, Margaret C—, aged 10, was admitted as my patient into St. George's Hospital, though my absence from town prevented my noting her later symptoms from

personal knowledge. She had been attacked with acute rheumatism a month before, of which only the cardiac results were now apparent. The apex was lower than natural, and marked the position of a loud systolic murmur. She had no palpitation or dyspnœa, though there was some pain in the left side of the chest on deep inspiration, and pain also in the spinal'region, which, however, as yet, did not receive much notice. Under small doses of iron and digitalis she seemed to improve, and on the 19th was able to leave her bed. When up, slight choreic movements were for the first time observed. These, slight as they were, terrified the child. A little friend of hers had lately died of chorea; she had watched and imitated the movements, and when she recognised them in her own person, made sure that she should meet the same fate. She was perturbed in mind, restless, and sleepless. On the 21st the movements much increased, and at the same time a little blood was found in the urine. In the evening she was seized with acute pain which she referred to the lumbar region of the spine, and thought to be relieved by rubbing.

When this had lasted about an hour she somewhat unexpectedly died.

On post-mortem examination, about half a pint of clear fluid was found in each pleura, and two ounces in the pericardium. The heart was increased to the weight of eight ounces; it was partially contracted; the free edge of the mitral valve was thickened and on its auricular surface was a fringe of small fibrinous beads, easily detached; similar vegetations were found upon the edges of the aortic valves. The liver, spleen, and kidneys were tough and congested. No other morbid appearances were discovered save in the nervous centres. The brain was congested in membranes and substance. The puncta vasculosa were marked, and from many of the vessels of the oval centres and the medulla threads of black coagulum could be drawn. The spinal cord, in the superficial examination which was made in the fresh state, was not noted as unnatural.

Several parts of the brain, which it is not necessary to describe in detail, were examined in section with results such as in some of the cases related subsequently, though of earlier occurrence, are described more minutely.

The vessels were loaded with blood, those of the size above the smallest the most remarkably so; arteries and veins were both affected; when one only, then generally the vein. The corpora striata and the arbor vitæ were thus markedly affected; in the latter were one or two considerable holes which represented eroded perivascular canals.

In the cord were more unusual deviations. The central canal was dilated in a manner to which I have seen no parallel (see Pl. I, figs. 1—4). The enlargement was great in the cervical region, where the transverse diameter of the canal varied from $\frac{1}{30}$ of an inch to half as much; less in the dorsal, where it diminished to about $\frac{1}{60}$; greatest in the lumbar, where it reached a seventh of an inch, occupying exactly one third of the entire diameter of the cord. In the cervical and dorsal regions the cavity was mostly filled with granular blood-coloured material which evidently largely consisted of altered blood; in the lumbar region the canal was empty. In the dorsal region, where the contents of the canal were most abundantly sanguineous, the larger vessels were injected to the uttermost, distended and loaded veins lying in the grey matter on both sides of the canal and traversing the white columns.

In the upper part of the canal, particularly in the dorsal region, where the congestion was extreme, blood had been poured into it, and was of course found *in situ*; elsewhere the distending fluid had apparently been serum which had escaped when the cord was cut.

Beyond these changes, which may be briefly summed up as hyperæmia of the cord with distension of the canal by blood and serum, there were few others which call for notice. The anterior fissure, particularly in the cervical region, was eroded and contained blood-tinged effusion.

CASE 2.—Mary C— died at the age of ten years under my care at the Hospital for Sick Children in her third attack of chorea. The first, four years previously, was attributed to her having fallen into a pond; she was treated for it in the hospital and recovered under sulphate of zinc. The symptoms were then nearly as severe as in the last and fatal seizure. She was ill five months, but recovered perfectly. The second attack, two years afterwards, was slight and transient. After it she remained well for eighteen months, until on the 21st of May, 1874, she was butted by a goat which a boy had set at her, pushed down, and much frightened, though but little hurt. On the 25th her legs were observed to twitch, and later the arms. The movements daily increased, speaking and swallowing became difficult, she became sleepless and feverish, complained of pain in the lower and middle parts of the back and in the thighs, and on the 10th of June was admitted for the last time. She had never had rheumatism.

When admitted the symptoms were severe; the movements of the limbs were so violent and continual that splints were required for

their restraint—a measure nowise objected to by the child. There was much difficulty in articulation, but none in the choice of words. Her words were correctly selected, though uttered interruptedly, with much effort and grimace, and often only in a whisper. There was some difficulty also in swallowing, due apparently to the disturbed action of the mouth and tongue. She had sordes on the lips, a dry tongue, and much febrile disturbance, with a temperature gradually increasing up to 104·5. A faint systolic murmur was audible at the apex. She was sleepless and the bowels confined; conditions to the obviation of which the early treatment was mainly directed.

On the night of the 11th, having been but three days in the hospital, while in the act of taking some brandy and water she suddenly collapsed, became blue, and died. In the early part of the night she had slept from chloral.

On post-mortem examination it was found that, putting aside the nervous centres, all the organs were healthy except the heart. The pericardium was natural, the mitral valve only affected. Along its auricular surface, just above the free edge, was a straight regular line of small close hard vegetations, all firmly fixed, and without adherent fibrine. The condition of heart was characteristic of the disease, just this amount of vegetation, without irregular fibrine, and thus limited to the inner surface of the mitral valve, being found in a considerable proportion of instances of death by chorea.

There was a slight excess of arachnoid fluid, but none in the ventricles of the brain. The grey matter was dark, the red points of the white numerous. The microscope showed a generally scattered loading of the vessels up to about $\frac{1}{50}$ of an inch in diameter, both arteries and veins. Distension with blood persistently after death is obviously more distinctly unnatural in arteries than in veins; in this instance both were conspicuously affected. The large superficial veins of the corpora striata were distended to the utmost; and some of the larger arteries passing through its substance were surrounded (see Pl. I, figs. 5—7) by translucent, structureless, or delicately granular material, apparently an exudation of liquor sanguinis, lying between brain and vessel. In the pia mater and subarachnoid space were many extravasations of blood in toto; such were well seen in the anterior fissure of the medulla (see Pl. I, figs. 5—7), where large distended arteries were involved in a loose mass of corpuscles, within the meshes of the subarachnoid, which had apparently escaped, not by rupture, but migration. The brain substance was nowhere broken by the extrusion.

In the cord many still loaded vessels entered the grey matter;

there were no evidences of hæmorrhage, though many of perivascular change probably due to escape of at least the liquid part of the blood. In the dorsal and lumbar regions a large round hole full of granular and globular *débris* lay at each end of the commissure around the arterial branches which came from the anterior fissure. Excavations of this kind and in these situations are, it may be observed, common to many morbid states of the cord, as the results, apparently, of congestion and transudation. Less common perivascular changes were found in the lumbar region, where dilated arteries traversed the grey horns separated from their substance by a wide interval (see Pl. II, fig. 1) filled with the globular translucent matter which so often marks the contact of nervous tissue with dilated vessels. Transudation from the vessel and erosion thereby of the channel in which it lies would seem to constitute the major parts of the process, of which the results have been described.

CASE 3.—A girl, 7 years of age, died at the Hospital for Sick Children in a first attack of chorea, under the care of Dr. West, to whom I am indebted for the opportunity of examining the nervous centres.

There was no history of rheumatism or fright. The only ostensible cause for the illness was that she was wet through the day before the first symptoms appeared. The attack was severe though uncomplicated. Death occurred on the 24th day with signs of prostration which had come on rather abruptly. She had had but little sleep, and the movements had been violent and general. She had been treated chiefly with antimony.

On post-mortem examination all the organs were found to be healthy except the heart and the nervous centres. The heart had a line of small recent vegetations along the auricular edge of the mitral valve.

The sinuses of the dura mater were full of blood, some of which had coagulated. The surface and substance of the brain were also injected. Portions of this centre, with the spinal cord, were remitted to me for further examination.

The peculiarities of the brain may be summed up in the word injection—arterial, venous, and capillary. The smaller arteries and veins, whether in the superficial pia mater, in the fissures, or in the substance of the brain, had remained after death full of blood, which, solidified by the chromic acid, assumed the appearance of a general thrombosis. This loading of the blood-vessels affected all parts of the brain and all sizes of vessel; it was exceedingly marked in both arteries, and veins of about $\frac{1}{150}$ of an inch in diameter, and extended

intò the lesser ramifications until it reached the capillaries, many of which, even of the smallest size, were made conspicuous by the crowding of blood-corpuscles within them. The capillary injection was most marked in, though by no means confined to, the optic thalami. There were few evidences either of extravasation or of perivascular change; in one or two instances blood had escaped from the distended vessel into the surrounding sheath. The cerebellum, and conspicuously the neighbourhood of the dentate body, shared in the injection, particularly of the larger vessels.

The cord was congested, but to a less degree; a few large loaded vessels passed through the white into the central grey matter. In the dorsal and lumbar regions the grey matter showed the effects of hæmorrhage within its tissue. In certain tracts both of the dorsal and lumbar regions the grey matter had been broken or locally destroyed, with lateral symmetry, at the centre of each horn. The disruption at its greatest extent was nearly enough to cut the crescent through its middle and sever the anterior from the posterior cornu. The affected spot consisted of a somewhat irregular rending or crumbling of the grey matter, the broken-up nervous substance being more or less mingled with faded and altered blood, and the cavity fringed with the globular product of nervous disintegration. The result could only be ascribed to hæmorrhage some little time before death, simultaneously at several points in the grey matter, similarly disposed with regard to the two sides.

The morbid appearances in this case may be briefly described as those of injection of the brain and extravasation into the cord.

For the clinical facts of the following case and permission to examine the nervous centres I am indebted to my colleague Dr. Gee.

CASE 4.—Clara W—, aged 8, who had had scarlet fever five years before, but never rheumatism, was, while apparently well, frightened by a boy who had hidden himself in a dark room, and agitated much out of proportion to the cause. Three days later choreic movements showed themselves, and gradually increased until she was brought to the Hospital for Sick Children, fourteen days after their commencement. She then had violent choreic movements of the neck, trunk, arms, and legs. She could not stand or even sit without danger of falling. The face was little affected, but swallowing was difficult and speech hesitating. The embarrassment in the latter respect shortly increased so that she became scarcely able to utter her name intelligibly. There was no cardiac murmur, though the sounds were not perfectly regular.

She was placed on a water bed and treated mainly with chloral. The choreic symptoms much diminished, but she became prostrate, lapsed into the condition to which the term typhoid is applied, displayed some ulcerated patches about the fauces, and finally sank on the thirty-ninth day of her stay in the hospital, the fifty-seventh from the fright from which the illness dated.

An objection to the examination of the body was anticipated by the prompt action of the house surgeon, Mr. Parker, as far as the brain and cord were concerned, and to him I am indebted for being able to examine the cord and portions of the brain, as well as for the use of sections which he himself prepared.

The appearances of the corpora striata did not indicate any changes beyond hyperæmia. The veins were mostly loaded with blood, the large superficial vein, which was similarly affected in the case of Mary C—, No. 2, conspicuously so. The injection was more marked in the veins than the arteries; in some cases where vessels of each sort lay together, the vein only was full.

There was no evidence of ante-mortem coagulation, of extravasation either of corpuscles or liquor sanguinis, or of erosion of tissue. Some empty dilated veins were seen which apparently had been loaded to distension at no remote date.

There were also evidences of hyperæmia in the medulla, which increased on reaching the cord and attained their climax in the cervical enlargement, where was a large mass of extravasated blood (see Pl. III, fig. 1). This, which was obvious enough to the naked eye, lay in a torn cavity within the left horn, involving the outer margin of its central and posterior portion. The clot in transverse section measured $\frac{1}{15}$ of an inch long, and a third as much in width. The blood had evidently been effused long enough before death to have undergone change; though still characteristic in colour, it had lost its corpuscular structure.

Beside the large mass described there was evidence of hæmorrhage —blood-coloured exudation mixed with products of the disintegration of adjacent surfaces—in the anterior fissure, the central canal, and one of the arterial channels of the commissure.

Some of the vessels of the cord of both kinds, more especially in the lateral columns and posterior horns, were distended. Some were strikingly so in the right posterior horn, in the spot corresponding to the hæmorrhage on the other side. Distended vessels were seen in the rest of the cervical region, and, indeed, to a less extent, throughout the whole cord. Vessels, chiefly venous, irregularly distended often to the utmost, were frequent in the white matter impinging upon the lateral aspect of the grey. These were

numerous in the dorsal region, but there was no hæmorrhage save in the cervical.

Perivascular erosions, in which the products of destruction of tissue were mingled with those of exudation, were found in the lower portion of the medulla and in every region of the cord. These were placed chiefly at the bottom of the anterior fissure, and continuously with it in the transverse commissure. In this case, however, the destruction of tissue was less marked than hyperæmia and extravasation.

CASE 5.—John P—, aged 11, came under my care in St. George's on the 30th of December, 1874. Six weeks previously he had been attacked with acute rheumatism the articular pains of which lasted three weeks. They were immediately succeeded by a habitual swinging of the arms and legs, which when remarked upon the boy said was "for exercise." Twitchings of the facial muscles ensued, with jactitating loss of command of the limbs and nearly complete loss of speech, apparently from difficulty in the articulation rather than in the choice of words. He had a loud systolic murmur at the apex, with evidence of hypertrophy of the heart. Under the valerianates of zinc and iron the choreic movements lessened, the use of the limbs and the power of speech were slowly restored, and on the 27th of January he was sent to Wimbledon as convalescent.

There the heart symptoms became more pronounced without any return of those of chorea. He had pain in the chest, blueness of the lips, and intolerance of the horizontal posture. In the night of February the 12th he expired with signs of cardiac distress.

On post-mortem examination it was found that the surfaces of the pericardium were universally adherent partly by old and partly by recent lymph. The free edge of the mitral valve was fringed with a row of fibrinous beads, which were most abundant upon its auricular surface. The edges of the aortic valve were similarly fringed. The lungs were slightly congested and œdematous, and the bronchial membrane vascular. All the other organs, including the brain and cord, were natural to the naked eye.

Subsequently sections from almost every region of the brain were examined microscopically. They were in most instances natural, the nerve-cells invariably so. Save some injection of the vessels not enough to be decidedly morbid, though the veins were much distended in particular about the dentate bodies of the cerebellum, the vessels and their canals were normal. There was no extravasation, effusion, or erosion.

In two situations, however, were remarkable exceptions to these statements.

In the deeper white matter of one of the cerebral convolutions were many conspicuous spots which consisted, as represented in the drawing, of accumulations of crystals of hæmatine (see Pl. II, figs. 2, 3), mingled with indefinite *débris* probably of nervous origin, swelling the canals around arteries which still remained distended with blood.

The other region referred to as the seat of significant change is that of the corpora striata. These bodies were more minutely injected than the rest of the brain, the capillaries, as well as the larger vessels of both classes, being packed with blood-corpuscles; and numerous spots, striking objects under the microscope, were closely set in their substance. These consisted each of an artery in section, empty, crumpled, and collapsed (see Pl. I, fig. 6), and surrounded with a mass of globular *débris* which had been formed at the expense of the surrounding tissue. They had evidently been produced by a solution or destruction of tissue around the vessel, consequent upon effusion from it, the result of injection, which had now ceased to exist. In time these mixed effects of extravasation and disintegration would have disappeared and left mere vacuities.

The spinal cord displayed loaded vessels and eroded fissures, such as were seen in every other instance examined. In addition to these common changes the grey matter had undergone extensive transformation of the kind to which the term *sclerosis* has been given. This was slight in the cervical region, extreme throughout the dorsal, absent from the lumbar. The change was confined to the grey matter, which it affected on both sides of the cord, nearly symmetrically (see Pl. II, figs. 2, 3). In the dorsal region it involved at least a third of the grey matter, as seen in section ; the affected portions on each side being adjacent to the attachment of the transverse commissure and at the root of each posterior horn. In the cervical region, though the change was less extensive, its position was the same. The altered grey substance had become converted into a wool-like entanglement of curving areolar fibres among which nerve-cells could be sometimes traced, especially near the edges, but from which all other nerve elements had disappeared, leaving a mere confusion of connective tissue. The nuclei proper to the healthy structure were present, but had undergone no increase, nor was there any other evidence of fibroid or connective new growth. The change seemed to consist essentially of a destruction

and removal of the nervous elements, their fibroid skeleton only remaining. This was best displayed in glycerine.

CASE 6.—On the 16th of March, 1874, Louisa W—, aged 13, came into St. George's, under the care of Dr. Ogle, with slight general rheumatism, affecting chiefly the knees and ankles, which had begun with a rigor six days previously. She had a loud systolic murmur at both base and apex, and slight movements characteristic of chorea. It was learned that she had on two previous occasions, the date of the earlier not recorded, the later a year ago, had chorea together with rheumatism, the choreic in each instance having distinctly preceded the rheumatic affection. Her symptoms in the final attack were not apparently threatening, and the chorea in particular obtained little attention. She died suddenly in the night on the thirteenth day of her last attack, the seventh of her stay in the hospital. The treatment had chiefly consisted of small doses of citrate of potash.

On post-mortem examination the mitral valve was found to be thickened and narrowed, while a line of soft fibrinous beads traversed its auricular surface near the free margin. The aortic valves were thickened and puckered, and to a roughened spot some nodules of fibrine were firmly adherent. The left ventricle was contracted; the right contained firm, partly decolorised clot. The pleuræ each contained about a pint of clear fluid, the pericardium an ounce. The kidneys were congested; all the other organs were natural save the nervous centres. The membranes of the brain and cord were congested; the puncta vasculosa of the brain were large and prominent; no other evidences of disease were apparent to the naked eye. Mr. Warrington Haward, to whom I am indebted so far for the post-mortem observations, kindly preserved for me the brain and cord in view of a more minute examination, of which I will now epitomise the results.

These were of two kinds—recent injection and its consequences belonging to the last attack, and ancient changes due probably to congestive processes associated with one of the earlier.

Beginning with the brain and taking the recent changes first, there was, especially between the floor of the lateral ventricles and the base of the brain, a remarkable injection, most conspicuous in the veins and capillaries, but sometimes involving also the arteries. The capillaries were exhibited diagrammatically by the blood-corpuscles within them. Large veins, in the optic thalami especially, remained thus obstructed and irregularly swollen, and in several instances blood-corpuscles had escaped both from veins and arteries into the

surrounding sheath. Kindred but somewhat older changes, evidently proceeding from such beginnings to the yellow spotting of so-called sclerosis, were numerously found between the floor of the ventricles and the base of the brain, more especially about the inner end of the left Sylvian fissure. Minute distended vessels, chiefly arterial or capillary, were here and there seen as the centre of a spot of colourless degeneration rejective of carmine, in which the nervous tissue had become transformed into a congeries of delicate "soap-bubble" globules sparsely intermingled with extruded blood-corpuscles. These were obviously an early stage of the striking form of sclerosis to be presently mentioned.

This, occurring in the same neighbourhood and manifestly but a later result of the same process (see Pl. II, fig. 4), took the shape of a very conspicuous sprinkling of the grey matter with cream-coloured spots which gave a piebald character to the section. The spots cut across were mostly circular, cylindrically elongated. They were often about $\frac{1}{100}$ of an inch in diameter, sometimes larger, more often smaller. They were closely placed; more than fifty were counted within the circumference of a sixpence. Their absolute refusal of carmine made them striking objects in the deeply tinted grey matter in which they lay. They were related in position to vessels; in several instances they were numerously disposed along the banks of a channel which contained an empty artery and a distended vein. The highest powers which could be brought to bear upon the spots failed to display any more definite structure than a dim globulation akin to the more distinct texture of the perivascular degenerations previously described. The change could not be otherwise regarded than as a circumscribed degeneration of the grey matter determined by some vascular conditions, which no longer existed, probably dilatation and effusion. The part of the brain thus affected, which was very small in extent, was identified as belonging to the left middle lobe and the neighbourhood of the Sylvian fissure, apparently to the substantia perforata and the ascending parietal convolutions close to the fissure of Rolando.

The spinal cord was traversed throughout by enormously swollen vessels which were mainly venous, and were largest and most numerous in the dorsal region. Veins thus full and swollen occupied the lateral white matter and impinged upon the central grey, which itself was minutely injected, more especially in the posterior horns. Beyond these changes there was much erosion, especially in the cervical region at the bottom of the anterior fissure and in the course of the vessels in the commissure. In the same region a spot

of recent destruction of the grey matter, allied to those found in the brain, existed in contact with a distended artery.

Case 7.—Mary O—, a widow, aged 54, came to me as an out-patient on the 23rd of July, 1873, and was at once admitted under the care of Dr. Fuller. She had the symptoms of chorea in a violent form. The movements of the head, face, and upper extremities were extravagant and with little interruption; the face was in constant contortion and grimace; the left arm was somewhat more affected than the right; the lower extremities but little. She had much difficulty in putting out and keeping out the tongue, and in the articulation of words. These muscular disturbances were all increased by mental agitation. She was thin, worn, and haggard. The sounds of the heart were natural. The urine contained a trace of albumen. The movements had come on four years before without determined cause, two years subsequently to the cessation of the catamenia. There was no history of either rheumatism or fright. The condition of exhaustion in which she was admitted increased; difficulty of swallowing was superadded to the symptoms already mentioned; and she sank after having been in the hospital for a fortnight. With the increasing prostration the choreic movements diminished.

On post-mortem examination it was found that all the organs were practically natural, save only that the kidneys were somewhat shrunken and granular. The heart was perfectly healthy. The nervous centres displayed no signs of disease to the naked eye, but with the microscope extensive and striking changes were discovered of the same kind and in identically the same situations as in the instance last described. A thick sprinkling of "disseminated* sclerosis" permeated the basal parts of the brain, more especially those fed by the middle cerebral arteries.

The regions most thickly beset were those known as the substantia perforata, lying between the base of the brain and the corpora striata, and giving passage to numerous small arteries which proceed from the beginning of the middle cerebral to those bodies. In the grey matter of this region a space, most of which might have been com-

* I have employed the term sclerosis in deference to general usage; but if it be held to imply induration from increase of connective tissue it is not appropriate to the condition here described, which is apparently one of de-generation of the nervous elements rather than increase of the connective. The conspicuous appearance of the latter in the affected parts of the cord was probably due to the destruction of the proper nervous structures, the fibrous skeleton of the tissue remaining.

prised within the circumference of a shilling, was closely beset with the characteristic spots. These were mostly circular or ovoid, and of a general diameter not far removed from $\frac{1}{100}$ of an inch. They sometimes lay on the banks of vessels, but were more often without immediate connection with them. They were less translucent than the surrounding healthy tissue, yellow, rejective of carmine, and faintly globulated in structure.

Though here, perhaps, most abundant, the changes were by no means limited to these regions. They were scarcely less numerously scattered in the grey matter of the corpora striata under the ventricular surface, and were frequently present in the whole region between the floor of the ventricles and the base. The two sides were affected with remarkable symmetry; a section transversely through both corpora striata showed the spots of sclerosis in each, in almost precisely the same situations—chiefly in the superficial grey matter along its junction with the white.

There was little else to note, save that the arteries, particularly where these changes abounded, were loaded with blood, with here and there signs, slight and seldom, of perivascular disintegration.

The cord in the cervical and upper dorsal regions was distorted and torn by the intrusion into its substance and fissures of masses of translucent matter. These pools of exudation, as they may be regarded, presented themselves with much regularity in certain situations; one in the anterior fissure, one smaller and less constant in the posterior fissure, and a large one within the grey matter of each posterior horn. Beside these were others, smaller in size and various in position. The anterior fissure in the upper part of the cervical region was as shown in the drawing (see Pl. III, fig. 2) filled from top to bottom, and to distension, with material for the most part structureless, though fringed with nervous detritus. At the bottom of the fissure the commissure was in some places deeply eroded. The pools in the posterior horns, which were evident to the naked eye by their size and transparency, were so disposed that a line connecting them would have fallen but little behind the commissure. They were situate, that is, towards the bases of the posterior horns, precisely in the spots which in some of the earlier cases were the seats of hæmorrhagic extravasation, and in one of the later of so-called sclerosis. The masses in question, though often bordered by the products of nervous decay, had evidently resulted, not from transformation of tissue, but intrusion into it. They were generally in the position of vessels, though these were not now congested; in some instances the structure of the cord was obviously broken and displaced by them; their bulk was in many instances as

structureless as glass; and it was sufficiently evident that they had resulted, not in the degeneration of tissue, but in the extrusion of liquor sanguinis.

These were not noticed below the middle of the dorsal region. There was no general congestion of the cord, nor any further changes beyond what have been alluded to.

The changes can be briefly summed up as so-called sclerosis of the brain, with exudation into the cord alike the products of vascular distension and escape.

To sum up the changes in the nervous centres, taking the seven cases as they ranged in duration from two days to four years, they amount to this :

CASE 1.—Two days. Injection of vessels of all classes in brain and cord ; most marked in the corpora striata and arbor vitæ, and in the dorsal region of the cord. Traces of erosion widely distributed. Hæmorrhage into, and distension by serum of, the central canal.

CASE 2.—Twenty-one days. Similar injection of brain, with the addition of superficial hæmorrhages, and exudation around the arteries of the corpora striata. Injection of cord, and periarterial erosion in the dorsal and lumbar regions, marked in the grey matter.

CASE 3.—Twenty-four days. Injection of the vessels of the brain of every class, most numerously about the optic thalami ; some extrusion of corpuscles. Injection of the cord and hæmorrhage into the grey matter of both dorsal and lumbar regions, symmetrical with regard to the two sides.

CASE 4.—Fifty-seven days. Injection of the brain, chiefly venous, and of the corpora striata. Injection and erosion of the cord, with large hæmorrhage into the cervical grey matter and smaller elsewhere.

CASE 5.—Sixty-four days. Venous injection of the brain, especially of the corpora striata, wherein were also periarterial exudations. Arteries in the convolutions near Sylvian fissure surrounded by blood crystals and *débris*. Injection and scattered erosions of the cord. " Sclerosis " of grey matter in both the dorsal and cervical regions placed with bilateral symmetry.

CASE 6.—Fatal attack thirteen days. Two precedent attacks (to one of which the older changes were apparently due), the last a year ago. Recent injection, such as in the other cases, of the bodies at floor of the lateral ventricles, and of the cord. Older changes, periarterial degenerations and scattered spots of "sclerosis" in the *substantia perforata*, and convolution at beginning of the left Sylvian fissure.

CASE 7.—Four years. Spots of "sclerosis" numerously set in the substantia perforata, and grey matter of corpora striata, symmetrically placed with regard to the two sides. In cord large exudations into grey matter and fissures, chiefly in cervical region.

Thus the changes throughout the series were remarkably constant in kind and place.

In kind they were all (allowing that sclerosis is so) directly connected with vascular disturbance. The injection was general to all the vessels, most marked in the arteries. When the sources of hæmorrhage could be determined they were always arterial; the degenerations were usually periarterial and the spots of sclerosis similarly placed. The first visible change would seem to be the injection or distension of the arteries, succeeded by extrusion of their contents to the irritation and injury of the surrounding tissue.

In place, the changes affected both brain and cord. Whether in the brain or the cord the changes on the two sides were generally, sometimes almost exactly, symmetrical, both being often affected at the same spot, in the same manner, and nearly to the same extent; and in instances where no such symmetry was obvious, a tendency to it was indicated, as for instance in the occurrence of vascular distension on one side corresponding to hæmorrhage on the other. The parts of the brain most amenable lay between the base and the floor of the lateral ventricles in the track of the middle cerebral arteries; the substantia perforata, the corpora striata, and the beginning of the Sylvian fissures.

Of the cord no region was exempt, but perhaps the cervical and dorsal regions were usually more affected than

the lumbar. With regard to the vertical or physiological subdivisions of the cord, these all, whether white or grey, shared in the vascular distension; this condition, however, was usually most marked in the vessels belonging to or in connection with the lateral part of the grey matter about the root of each posterior horn. And it is to be observed that this also was the chosen situation of the more definite and special changes, whether hæmorrhagic as in two instances, sclerose or exudatory.

Speaking generally, the chosen seats of the choreic changes are the parts of the brain which lie between the beginning of the middle cerebral arteries and the corpora striata—the parta perforata; and in the cord the central portion of each lateral mass of grey matter comprising the root of each posterior horn.

The nature and steps of the morbid process, hyperæmia, exudation, and its consequences, are open to view, but not so the causes in which the series has taken origin. Arterial repletion seems mainly concerned in the development of the disease; why or at what bidding do these vessels thus gorge themselves ?

To seek the origin of the process we may fancifully attribute the hyperæmia to causes of two kinds, the first belonging to the blood, the second to the tissue. Irritants may be introduced with the blood either solid as emboli or liquid like the hypothetical fluent poisons of disease. Or the irritation may be of nervous origin, and the vascular dilatation secondary thereto.

The nature of the lesions in the brain and cord is not consistent with the somewhat attractive hypothesis of embolism. The lesions are indeed determined in position by the course of arteries, in the brain notably by the middle cerebral—favourite routes of emboli—and the perforating branches which pass from these to the corpora striata ; but in none of the instances described were decolorised fibrine, detached clots, or signs of impaction detected, and the erraticism of embolic accident was wantihg ; the constancy indeed with which the changes repeated them-

selves in certain positions and the equality with which they affected both sides of the body are conclusive objections to this hypothesis. The corpora striata for example were affected with almost absolute symmetry, notwithstanding that these bodies receive their blood respectively from the right and left carotids, and different parts of the aortic arch.

Observations of the concurrent circumstances of chorea may possibly assist us in understanding the nature and correlation of its lesions.

The salient facts of the disease—those which must be fitted together in any rational view of its nature—are these; its origin in rheumatism; its no less frequent origin, without rheumatism, in circumstances acting on the mind; and its constant association whether it has begun in one cause or the other with mitral endocarditis.

I have tabulated the particulars of twenty-two cases, examined after death at St. George's Hospital, and at the Hospital for Sick Children; representing the experience of thirty-three years at the one hospital, fifteen at the other.

PATHOLOGICAL

Particulars of 22 fatal cases. Those cases the names of which

Reference.	Name.	Age.	Previous attacks.	Cause of present attack.	Its duration.
Nov. 19— 1842	Emma L—	17	No history given	—	—
1844	Mary K—	15	Two	Amenorrhœa for 3 months before	3 mos. and 3 wks.
1845. No. 10	Mary H—, M.	26	Jactitation 4 years before, after confinement	Chorea after rheumatism; has had menorrhagia	3 wks.
1850. 113	George S—	19	Rheumatism before but not chorea	Rheumatism came on 4 days before chorea, slight, only of knees	10 dys.
1855. 309	Mary W—	17	None mentioned	No fright; amenorrhœa ?	9 dys.
1860. 295	Mary R—	7	None mentioned	Fright; pushed into a ditch	7 wks.
1863. 132	Jane G—	16	None mentioned	Attributed to fright; has had rheumatism, not acute	3 wks.
„ 167	Mary C—	15	None mentioned	Fright; shouted at in sleep; ? uterine irritation; catamenia irregular, once in last 7 months	?

TABLE.

are printed in italics are related also in the substance of the paper.

Rheumatism.	Other diseases.	Heart.	Pericardium.	Blocks or other evidence of embolism.	Other organs.
—	—	Healthy; valves healthy	Healthy	—	Ovaries large; uterus congested; bowels loaded; brain and cord congested.
No rheumatism ever	Caries of clavicle	Minute rounded vegetation on auricularsurface of mitral	Healthy	—	Uterus, vagina, and ovaries vascular.
Acute rheumatism 2 mos. before death; 10 days after recovery chorea began	Headache	Slender fringe of vegetation on auricularsurface of mitral valve	Healthy	—	Old adhesions between the spleen and diaphragm; uterus large and hard; os ulcerated; old adhesions about ovaries.
Rheumatic fever at 12 years of age, and also since more than once	—	Row of beads of firmly organised lymph along auricular surface of mitral valve (recent ?)	Firmly adherent (old)	—	Brain congested; cord congested, diffluent opposite the third or fourth upper dorsal vertebra.
None mentioned	None mentioned	Heart healthy; no murmur mentioned	Healthy	—	Cord "appeared" softened in dorsal and upper cervical regions.
None mentioned	Abscesses in chestwalls, &c.	Heart healthy; sounds natural	Healthy	—	Brain pale; abscess in chest-wall full of blood consequent on movements, &c.
Two months before chorea, rheumaticpains, redness of joints, and amenorrhœa	Pneumonia	Recent vegetations on mitral and aortic valves	Old lymph on pericardium	—	Brain and cord congested; uterine organs injected; early pneumonia.
None mentioned	? Masturbation or fornication	Healthy; no murmur	Healthy	—	Intense injection of the brain and cord; vagina and os bathed with pus; vagina dilated; os uteri widely open.

Reference.		Name.	Age.	Previous attacks.	Cause of present attack.	Its duration.
1864.	249	Leopold L—	11	None mentioned	No cause assigned or to be discovered	5 mos.
1867.	118	Harriet S—	17	None mentioned (unmarried)	Pregnancy; 4 months, disease coincident nearly with its beginning	4 mos.
„	305	Mary G—	12	None mentioned	No cause assigned	13 wks.
1869.	11	Catherine T—	15	None mentioned	Excitement (mental or uterine ?); catamenia present for the first time on admission	2 wks.
1874.	89	*Louisa W—*	18	Two previous attacks of rheumatism and also of chorea — chorea in each having preceded rheumatism	Slight general rheumatism several days before the chorea begun	13 dys.
1875	48	*John P—*	11	None	Succeeded upon acute rheumatism, after he had it for 8 weeks and apparently recovered	9 wks.
1873		*Mary O—*	54	None known (widow); no fright or rheumatism	Unknown; catamenia ceased 2 years	4 years
1875		*Margaret C—*	10	None known	Acute rheumatism, begun 5 weeks before chorea	—
Ch. Hosp.	97	Maria P—	11	One	History imperfect; probably connected with rheumatism (pericardial adhesion)	2 mos.

Rheumatism.	Other diseases.	Heart.	Pericardium.	Blocks or other evidence of embolism.	Other organs.
None mentioned	—	Line of beads of lymph on inner edge of mitral valve (auricular)	Healthy	—	Cord and brain injected; grey matter, altered yellow in patches.
None	Worms, pneumonia	Mitral valve slightly thickened, and on its auricular surface some fibrinous deposits, evidently quite recent, could be pulled off	Healthy	Fibrinous block in left kidney and in liver	Brain congested; pneumonia; one round worm in duodenum.
"No history of rheumatism obtained"	Pneumonia	Minute beads of fibrine on auricular aspects of both mitral and tricuspid valves, also on endocardium; all quite recent	Healthy	—	Pneumonia; brain wet, clots in vessels at base, but not decolorized or apparently abnormal in any way.
None	—	Beads of lymph on auricular surface of mitral, soft and fresh (no murmur)	Healthy	—	Brain highly injected; lungs congested; old tubercle in spleen; ovarian cyst; signs of recent menstruation.
Rheumatism with each attack of chorea	Hydrothorax	Old and recent disease of mitral and aortic valves; rough murmur during life	Little fluid in pleuræ	—	Fluid in pleuræ (see notes of brain, in text, p. 10).
Three wks. of acute rheumatism, on cessation of of which movements began	—	Mitral and aortic valves fringed with recent fibrine	Pericardium adherent	—	Lungs congested (see p. 8).
None	Trace of albumen	Healthy	Healthy	—	Kidneys granular (see p. 12).
Acute rheumatism absent when chorea begun, cardiac results only present ?	—	Recent vegetations on aortic and mitral valves; heart hypertrophied	Healthy	—	Dilatation of cord, &c. (see p. 2).
	Diphtheria	Murmur not evident after first 2 weeks; recent vegetations on mitral and tricuspid valves	Old adhesions	—	Diphtheria.

Reference.		Name.	Age.	Previous attacks.	Cause of present attack.	Its duration.
„ v. 2.	239	Athalia W—	9	None ; chorea, chronic, confined to left side	None known ; scarlatina (caught in hospital) 3 weeks after admission, rheumatism on this, then diphtheria	20 dys.
v. 3.	28	*Amelia P—*	7	None	Wet through day before attack began ; no rheumatism or fright	21 dys.
„	136	Ruth J—	10	None mentioned	? Stiff neck	4 wks.
„	182	Mary A. H—	8	5 or 6 attacks, first apparently caused by fright, but preceded by rheumatism 4 months before ; fright, fell into ditch	Mental excitement ?	—
„	227	*Mary C—*	10	Bad attack of chorea (from fright, fell into pond) 2 years before ; had rheumatism, slight attack 18 months ago	Frightened by a goat	15 dys.

Rheumatism.	Other diseases.	Heart.	Pericardium.	Blocks or other evidence of embolism.	Other organs.
Came on for first time after scarlatina caught in hospital	Scarlatina; rheumatism 4 days afterwards; diphtheria; tracheotomy	When admitted no murmur; afterwards systolic murmur, with rheumatism; granulations on auricular surface of mitral valve	Healthy	—	Results of diphtheria.
None	—	Murmur came on subsequently to chorea; minute vegetations on auricular surface of mitral valve	Healthy	—	See p. 5.
Stiff neck 4 weeks before chorea	Pleuro-pneumonia	Enlarged; slight fibrinous deposit on aortic and mitral valves	Friction when admitted extensive; lymph in pericardium	—	Scattered tubercle, some in pleura; recent pleurisy.
Acute rheumatism 4 months before first attack, none afterwards	Pleuro-pneumonia	Slight fibrinous deposit on all four valves	Pericarditis came on at Highgate, subsequently to chorea, endocarditis of all the 4 valves, slgt.	—	Pleurisy (came on at Highgate); pneumonia; liver and kidneys congested from cardiac lesion.
None	—	Systolic murmur not present until 3 days after admission; line of small recent vegetations along auricular surface of mitral, other valves natural	Healthy	—	See p. 4.

Abstract of preceding Table, showing the distribution of Heart Disease among the subjects of Chorea, classed according to the apparent Origin of the Chorea.

Origin of chorea.	Healthy.	Recent vegetations on mitral valve only.	Recent vegetations on mitral, with old thickening.	Recent vegetations on mitral and aortic.	Recent vegetations on mitral and aortic, with old thickening of both.	Recent vegetations on mitral and aortic, with pericardial adhesions.	Recent vegetations on mitral and tricuspid.	Recent vegetations on mitral and tricuspid, with pericardial adhesions.	Recent vegetations on mitral and aortic, with recent pericarditis.	Recent vegetations on mitral, with old pericardial adhesions.	Total number of cases affected with recent endocarditis.
Rheumatic (6 cases)	...	1	...	1	1	1	...	...	1	1	6
Mental (2 cases)	1	1	...	...	...	...	...	...	...	...	1
Uterine (3 cases)	1	1	1	...	...	...	...	...	...	...	2
Rheumatic + Uterine (1 case)	...	1	...	...	...	...	...	...	...	...	1
Mental + Uterine (2 cases)	1	1	...	...	...	...	...	...	...	...	1
Rheumatic + Mental (2 cases)	...	...	...	...	...	1	...	...	1	...	2
Unknown (6 cases)	2	2	...	...	...	...	1	1	...	...	4
Total (22 cases)	5	7	1	1	1	2	1	1	2	1	17

The almost invariable association of endocarditis at least with the chorea of childhood, at once becomes prominent, and with it the pathological difficulty of the disease, the connection between the nervous and the cardiac disturbance, presents itself.

I subjoin an abstract from the preceding table which will show at a glance the frequency of endocarditis with chorea, be the cause of the chorea what it may.

Thus of twenty-two fatal cases of chorea the heart was found to be healthy but in five; and of these five exceptional cases only one was a child. In every instance making up the large tale of cardiac disease, there were recent vegetations on the mitral valve, and often also elsewhere. Thus in childhood, at least, endocarditis is an almost invariable accompaniment of fatal chorea. Evidence of present or past pericarditis was found in six of the number; a large proportion due no doubt to the frequency of rheumatism in association with chorea. The cardiac characteristic of chorea, however, is not peri- but endo-carditis; or at least beading by lymph especially of the mitral valve. Why are the valves thus constantly affected? is a question which at once presents itself. Does the endocarditis cause the chorea by embolism or otherwise? does the chorea cause the endocarditis or what passes for it? or are the two disorders the independent results of a common cause? These questions are sufficiently intricate to demand a somewhat careful examination of the circumstances in which the concurrence is found.

Rheumatism is known to be a frequent antecedent of chorea; it must be asked first of all how often the endocarditis of chorea is rheumatic. Taking the seventeen cases of choreic endocarditis in the table, and looking at the antecedents of the disease in each instance we find three in which there was no clue as to the cause of the disease. Putting these aside we find six in which the disorder was apparently due to rheumatism; three in which pains probably rheumatic had occurred at some

time, though possibly other circumstances were more con-
cerned in the production of the chorea; one in which an
adherent pericardium without any other record of rheu-
matism was accepted as sufficient evidence of its occur-
rence. Thus it was not possible by any presumption to
associate rheumatism with more than ten of these cases;
leaving four in which the disorder was traced, to the
definite exclusion of rheumatism, to mental or uterine dis-
turbances, or a conjunction of the two. And so far the
facts point to the inference that there is a pathological
link between chorea and endocarditis with which rheuma-
tism has nothing to do.

To throw further light upon the connection of heart
disease with chorea, I have tabulated the clinical details
iu seventy cases, under my own care at the Hospital for
Sick Children, all minutely recorded by a succession of
competent registrars. The causes of each attack were
more fully traced than in the post-mortem series, but the
evidence relating to the heart is necessarily only stetho-
scopic. Thus the two series, the pathological and the
clinical, in some sort supplement each other.

CLINICAL TABLE.

70 non-fatal cases treated in the Hospital for Sick Children.

Cases dependent upon, or possibly connected with, rheumatism. Those most markedly so are placed first. Doubtful or equivocal cases are marked with an asterisk.

Name.	Age.	No. of attacks, if more than one	Cause or concurrent circumstances of each.	Rheumatism.	Heart.	Other diseases and remarks.
Herbert H—	5	...	Succeeded immediately upon acute rheumatism	Acute	Murmur at apex	
Amelia S—	13	...	Succeeded immediately, but gradually, upon acute rheumatism	Acute	No murmur	
Louisa H—	8	...	Immediately succeeded upon 3 days of slight articular rheumatism	Slight in several joints, 3 days	Loud systolic murmur at apex, nearly ceased at last	
Louisa N—	7	3	First attack.—Scarlatina followed by rheumatism. followed by chorea; fright: unknown	Slight rheumatism after scarlatina before first attack	The second attack, action irregular, soft systolic murmur at apex. In the third, action irregular, no murmur	
Joseph N—	8	2	First attack followed rheumatism; the second, mental shock: police-man came to say that his father was taken to hospital	Before first attack	Second attack only noted. At first natural; after a time the sounds became irregular, and slight murmur came at apex	
" "	10	3	Third attack two years after second, brought on by fright: bullock broke from slaughterhouse	None since first attack	In third attack the sounds weak, no murmur	
Ellen T—	10	...	Slight rheumatism in the knee just before	Slight local rheumatism just before chorea	Faint systolic murmur, most audible just above and to right of nipple	

Name.	Age.	No. of attacks, if more than one	Cause or concurrent circumstances of each.	Rheumatism.	Heart.	Other diseases and remarks.
Emily F—	9	2	First attack immediately after rheumatic fever; second without obvious cause	Acute rheumatism before first attack	Faint systolic murmur at base	
Ann B—	9	2	Rheumatic pains before first attack; cause of second uncertain	Rheumatic pains before first attack	In second attack natural; in first not noted	
Ann S—	8	...	Came on a fortnight after general rheumatism	Preceded, with an interval, by acute rheumatism	Irregular action, soft systolic murmur at base and apex	
Louisa S—	3	...	Chorea began 6 weeks after commencement of acute rheumatism	General articular rheumatism shortly before	Soft and slight systolic murmur at base	
Florence, N—	10	2	First attack followed scarlatina and rheumatism	Rheumatic pains preceded first attack, accompanied second	Rough systolic murmur at apex	
Eleanor B—	P	...	Pain in finger-joints before chorea began	In fingers before chorea began	Slight systolic murmur at apex	
Arthur D—	8	...	Came on 6 days after subsidence of general articular rheumatism	Acute, subsided 6 days before	Systolic murmur at apex	
Arthur B—	10	2	First came on after pains in joints; second after excitement at school	Rheumatic pains before first attack	Slight occasional murmur at apex in second attack; state of heart in first not known	
*Emily M—	9	4	First and second attacks after fright; cause of others unknown	Doubtful pain in ankles before first attack	Loud systolic murmur at apex and base	
William Q—	9	...	Mother and aunt insane; child restless, excitable, and subject to delusions; no ostensible cause	Slight in one joint after chorea had come on	No murmur, second sound sharp	

Name	Age		History	Antecedent condition	Cardiac signs	Remarks
*Jessie I—	7	2	First attack immediately preceded by "low fever" of 3 weeks duration; cause of second unknown	None? at beginning of second attack; complained of "something pinching her toes"	In first attack occasional systolic murmur at apex; in second ditto, with irregular action and doubtful old pericardial friction	Herpes
Henrietta D—	10	2	Father a lunatic; first attack fright; knocked down by a cab; second unknown	Attack of rheumatism between those of chorea	Action irregular; prolongation of first sound to right of nipple	Very severe
Mary W—	11	...	Not decided; began with pain in right arm and thumb possibly, but not apparently rheumatic	Doubtful pain in arm and thumb	Systolic murmur at apex and base	
*Charles C—	11	...	N ,probably — arm when it began to twitch	Probably none; pain in arm	No murmur, sounds irregular	
*Isabella J—	11	...	Father epileptic; two years ago had scarlatina, followed by rheumatism; chorea, of 5 weeks date only, preceded by irritability	Scarlatina and rheumatism 2 years ago; none in connection with present attack	Slight systolic murmur at apex; afterwards only occasional	
Henry B—	7	...	Not to be recognised	Slight rheumatism 9 months before	Systolic murmur at apex	
*Ellen G—	10	...	None recognised; choreic movements, preceded by pain in limbs; choreic?	Pain in limbs, probably choreic	Irregular, faint systolic murmur at apex, at base, and in vessels	
*Elizabeth G—	12	...	No immediate cause for chorea; two attacks of rheumatic fever, the last a year back	Two attacks of acute rheumatism previously	Action irregular, slight mitral murmur	
*Louisa M—	10	2	Indefinite pains in legs and arms, possibly rheumatic	Vague rheumatic pains	Systolic murmur at apex	
*Mary M—	10	...	Immediately succeeded very painful extraction of tooth; rheumatism after scarlatina 4 months before	Scarlatina 4 months before	Irregular, no murmur	
*Charles K—	10	...	Pains in limbs and shoulders at beginning of chorea (choreic?)	Doubtful pains in affected limb (choreic?)	Soft blowing murmur at apex	
*Caroline P—	9	...	Came on after "low fever"	Has had occasional muscular pains, thought to be rheumatic	Soft systolic murmur at apex, not constant	

Cases apparently entirely unconnected with rheumatism (from fright or other mental disturbance).

Name.	Age.	No. of attacks, if more than one	Cause or concurrent circumstances of each.	Rheumatism.	Heart.	Other diseases and remarks.
Arthur L—	8	...	Fright: chased by a cow; movements came on same night	None	Indefinite murmur at apex; subsequently ceased	Most severe
Emily E—	9	...	Frightened by drunken man; sleepless same night; illness immediate	—	Action irregular, no murmur	
Ann N—	7	...	Struck by her father when drunk; much terrified; chorea immediate	—	Action irregular, no murmur	
,, ,,	9	...	Second attack 2 years later; came on after being "smacked;" insanity in family	—	Action irregular, no murmur	
Annie S—	7	2	Much frightened by horse plunging; chorea immediate; cause of second attack not apparent	—	Natural with first attack; slight basic murmur with second	
Sarah E—	11	2	Repeatedly frightened by having to pass a certain dog; second attack also from fright	—	No murmur; intermittent pulse	Rickets
Georgina B—	9	...	Frightened by going into a room where was a corpse	—	Action irregular, no murmur	
Charlotte B—	9	...	Frightened by stories told her with regard to a hole in the ceiling; chorea immediate	—	Slight systolic murmur at apex, which went away	
Jeremiah C—	10	...	Father came home in the night drunk and greatly alarmed him	—	Occasional prolongation or slight systolic murmur at apex	
Matilda M—	9	...	Frightened by a dog	—	Sounds irregular and muffled; first sound almost with murmur	
James F—	11	3	First due to fright: nearly run over; cause of two other attacks unknown	—	Action irregular, soft systolic murmur at apex; diminished with cessation of chorea	
Richard B—	10	1	Fright: horse ran away; chorea began with partial paralysis same evening	—	No murmur	

Name	Age	Dur.	Cause		Heart sounds	Remarks
Alice B—	?	2	ng beaten	—	Irregular occasional murmur at apex, slighter at base	
Susan D—	10	2	of dog; cause of second	—	Action increased, but no murmur	
Matilda C—	8	...	fright some months uncertain	—	Irregular, slight prolongation of first sound	
Thomas M—	9	2	cause for first attack; cond	—	No murmur, second sound loud and reduplicated	
Elizabeth B—	6	...	by played a trick upon	—	Action irregular, no murmur	
Ann D—	9	2	ttributed to fright, but clusively	—	Action irregular, slight systolic murmur	
Emily S—	9	...	re beaten; immediately not twitch for a fort-	—	Murmur, with first sound at apex	Chorea ceased on access of severe toothache

Causes various or not to be ascertained.

Name	Age	Dur.	Cause		Heart sounds	Remarks
Emily C—	11	...	Hysteria? no other or more definite cause ascertainable	—	Natural	Hysteria
Henry J—	5	...	Thread worms?	—	Natural	
Alice H—	12	...	Congenital syphilis?	—	No murmur	
Emma K—	8	8	No rheumatism or fright to be traced; thread worms?	—	The first attack.—Sounds irregular when admitted, but no murmur; loud systolic murmur at apex afterwards appeared. The second attack.—Sounds irregular, but no murmur. The third.—Sounds natural	All attacks very severe
Eliza C—	10	...	Attributed to hard place; no other cause ascertainable	—	Slight systolic murmur at apex	
Emma K—	9	...	Cong		No murmur	Corneitis
Charles C—	9	...	Came art	but neither	—	Natural	
Henry P—	10	...	Of 4 no ostensible cause	sad chorea;	—	Loud murmur, with first sound at apex, afterwards diminished	Very severe

Other diseases and remarks.	Hysterical cough

The proportion of cardiac disturbance as displayed by this table is very great. In fourteen only of the seventy cases were the sounds natural. They were merely irregular in eleven; reduplicated or unnatural but still without murmur in three; while in forty-two of the cases valvular murmurs were found, accompanied in sixteen instances with marked irregularity of action. Thus the total irregularity reached twenty-seven, or more than a third of the whole. In the forty-two instances in which valvular murmurs were found, these were systolic in every instance; at the apex only, in thirty-four cases; at apex and base, in six; at base only, in two. In one instance pericardial friction of some standing was audible.

Thus mitral endocarditis as characteristic of chorea is no less evident clinically than after death. It is to be remarked, however, as of common experience in chorea, that the murmur though mitral and organic is not always persistent; there are in this series six examples to the contrary. The evanescence of the murmur is no doubt due to the small amount of lymph usually deposited, and the nearly complete recovery possible to the valve. Passing from the murmurs of chorea in general to a particular consideration of their frequency in relation to its source, I append an abstract of the clinical table.

Clinical Condition of the Heart in Chorea as produced by different Causes (in 70 cases).

	Valvular murmur.	Sounds irregular or unnatural, but without murmur.	Sounds natural.
Association with rheumatism, ascertained or presumed (28 cases).			
Succeeded definite rheumatism (13 cases)	11		2
Rheumatism at connected with chorea (4 cases)	3	1	
First attack from rheumatism, second attack from fright (2 cases)	2		
Has had rheumatism: parent insane (2 cases)	1	1	
I the immediate cause; parent insane or epileptic (1 case)	1		
I choreic, or rheumatic? (5 cases)	4	1	
Chorea succeeded upon getting wet (1 case)			1
No rheumatism known to have existed at any time (42 cases).			
Succeeded immediately and definitely upon fright; never rheumatism (16 cases)	9	6	1
Attributed to fright, but with less certainty; never rheumatism (3 cases)	2	1	
Immediately caused by fright; parent insane; never rheumatism (1 case)		1	
Associated with hysteria (3 cases)	1		2
,, ,, congenital syphilis (2 cases)			2
,, ,, thread worms (2 cases)	1		1
Cause unknown (16 cases)	8	3	5
	42	14	14

This statement shows, as did the post-mortem series, that whatever be the origin of the chorea the heart is often—and it may be added is similarly—affected. Where the disease clearly succeded upon rheumatism, valvular murmurs were present in a large majority of cases; in twenty-two out of twenty-eight. Where it was as clearly produced by fright, without rheumatism, valvular murmurs were still present more often than not.

In twenty cases in which the chorea was attributed to mental causes, while careful inquiry failed to elicit any history of rheumatism, there were eleven with valvular murmurs, and seven more with only irregularity of action. The circumstances in which the disease arose were in at least sixteen of the instances so definite, while the result was so immediate, that there was no room for doubt as to its veritable origin in fright. These facts would seem to show that endocarditis (for the mitral murmur of chorea, as post-mortem evidence shows may be taken as always endocarditic) is a consequence of chorea; since it is equally impossible to suppose that these hitherto healthy children were the subjects of endocarditis when frightened, or contracted it subsequently, independently of the resultant nervous disease.

At the same time it is equally certain that endocarditis when associated with rheumatism is a frequent predecessor of chorea, and thus the connection between chorea and endocarditis is duplex, and our comprehension of the disease must be imperfect until we can unravel the mystery of this double relation.

The first question which presents itself is this. Chorea often follows rheumatic endocarditis; is the chorea caused by the rheumatism or by the endocarditis? When chorea succeeds upon rheumatism, endocarditis whether set up by the rheumatism or the chorea is so frequently present, that to ordinary and even somewhat extensive observation it might easily seem to be a necessary link between the two. But cases occur, though comparatively seldom, in which the chorea has clearly originated in rheu-

matism, and yet the heart when the patient comes under ob-
servation is found to give natural sounds.[1] It has never
been my fortune to have watched such a case so closely from
its origin in rheumatism to its ending in chorea as to be able
to assert that no murmur was at any time to be heard; but
rheumatic murmurs are so often persistent that the absence
of one shortly after the attack renders it probable that the
heart may have escaped from the first; and I think it
may fairly be inferred from the evidence adduced that
rheumatism may cause chorea without the intercurrence
of endocarditis. And it may be further stated that there
is no evidence that endocarditis apart from rheumatism
has any share in the causation of the nervous malady.
The embolic theory contradicted as it is by the minute
pathology of the nervous centres is no less opposed by the
rougher observations which the tables record. In the
post-mortem series of twenty-two cases there was but one
in which blocks or any of the recognised consequences of
embolism were found ; and it needs no large experience of
chorea to teach that its prevailing cardiac lesion—beading
of the mitral valve, fine, close, and regular—is not such as
is usually associated with the detachment and dissemi-
nation of fibrine. And to touch for a moment upon unre-
corded experience, it may be added that chorea is only
noticeable by its absence in cases where the occurrence of
embolism has been ascertained. I have never seen an
instance in which the well known blocking as found after
death had been conjoined in life with choreic symptoms.

Thus disconnecting, as we must do, chorea from mere
clot-scattering, we may go a step further and say as
experience warrants that heart disease except it be rheu-
matic is not an antecedent of chorea. Taking this with
what has been already shown, namely, that chorea may
follow rheumatism without disturbance of the heart's
sounds, we can but conclude that the chorea is caused by
the rheumatism, not by the endocarditis. The rheumatism,
in short, directly produces the congestive or sub-inflam-

[1] See cases in table of Amelia S—, Ann B—, and Charles C—,

matory condition of certain parts of the brain and cord which has been shown to belong to the disorder; or in other words chorea of rheumatic origin is rheumatism of the nervous centres.

Thus chorea and endocarditis may concur as the common but independent results of rheumatism; but that there is some other association between the two is evident from the frequent succession of endocarditis upon chorea, however disconnected from rheumatic antecedents. That in such cases the endocarditis succeeds upon the chorea is certain; it is clinically evident that the murmur does so; and the cardiac change when presented after death is of a recency corresponding to this view of its origin. How often non-rheumatic chorea is thus succeeded may be seen by reference to the single cause of fright; in sixteen instances resulting from this influence, definite and uncomplicated murmurs existed in nine, and mitral endocarditis, as we are justified in inferring, as often. The conclusion cannot be avoided that the cardiac is caused by the nervous disorder; the only doubt is how. The irregularity of cardiac action, by which choreic endocarditis is constantly preceded and accompanied, furnishes a suggestion in solution. It has long been observed that the muscle of the heart shares in chorea with other striped fibre. Of twenty-eight cases without murmur the action of the heart was irregular in eleven : of forty-two with murmur the heart was irregular in sixteen. Mere irregularity would seem to be the first change; irregularity with murmur the second. It used to be thought that the regurgitation of chorea was due only to muscular disturbance of the mitral valve; but the constant association according to post-mortem evidence of endocarditis with the murmur renders this explanation, to say the least, insufficient. But it may be suggested that regurgitation thus produced may possibly cause endocarditis, or, at least, the deposition upon the valve which passes for it. The beads are usually confined to the inner surface of the mitral valve, and arranged along the attachment of the

thin edge, where a line of minute but abrupt prominences
is presented to retrograde blood, but an arrangement of
more gradual slopes to blood flowing normally. Thus
possibly the collection of fibrine is the consequence not
the cause of the regurgitation.

Whether this explanation be, or be not the true one,
it must be held certain that chorea, be its origin what it
may, causes the valvular beading which is commonly re-
garded as synonymous with endocarditis; and it must be
believed that the cardiac has no share in the production of
the nervous disease, large as is the influence of rheumatism
in the relation.

It is not my purpose to dwell upon the causes of
chorea, excepting so far as they assist in the interpreta-
tion of its lesions; but it may be remarked that its origin
in the nervous system rather than as a vascular accident
is consistent with a predisposition to it which can be
recognised in bright and sensitive children, whose pink
and white complexions and colour of hair gives them what
is known as the Saxon type; and perhaps less markedly
in the descendants of the epileptic and insane.

To conclude then, we see in chorea a widely distributed
hyperæmia of the nervous centres not due to any me-
chanical mischance, but produced by causes mainly of
two kinds—one a morbid, probably a humoral, influence
which may affect the nervous centres as it affects other
organs and tissues; the other, irritation in some mode,
usually mental, but sometimes what is called reflex, which
especially belongs to and disturbs the nervous system,
and affects persons differently according to the inherent
mobility of their nature.

Given the irritant, mental, reflex, or rheumatic, the
course of the disease has been sufficiently traced in hyper-
æmia and its results. The spots of perivascular change
are widely scattered throughout that large region which
lies inferiorly to the cerebral convolutions between the
corpora striata and the lower end of the cord—the dis-

trict of the motor and sensory as distinguished from the mental functions. The result chiefly in muscular excitement, rather than in paralysis or loss of sensation (though it is to be observed that a lesser degree of both is frequently present), may be associated with the character of the lesions which are points of irritation rather than planes of section, and as such calculated to produce irritative rather than paralytic effects; not so much to cut off as unnaturally to excite nervous function. In looking at the nature of the lesions, proceeding as they do from vascular distension to perivascular change, it is not possible but to connect them with those of a large group of nervous disorders and prominently with those of diabetes. In diabetes the changes are similar in their origin and character, though usually seen at a later stage and when attended with more excavation. They are, however, somewhat differently disposed; in both they are largely distributed; in diabetes the stress falling widely upon the brain, markedly though by no means solely upon the medulla; in chorea, to the avoidance of the medulla, upon the region between the corpora striata and the base of the brain, and upon the whole tract of the cord.

Every period of life has its own regions of nervous susceptibility; in childhood the motor; in adolescence the emotional; in advancing years the mental, and coævally or nearly so, that part of the nervous mechanism which instigates glycosuria. Much the same mental impression may make a child choreic, a girl hysterical, or a man diabetic. And thus both in external origin, and in the nature though probably not in the site of the organic changes, we see resemblances and alliances between nervous disorders which in their symptoms betray little similarity.

DESCRIPTION OF PLATES I, II, III.

Dr. Dickinson on the Pathology of Chorea.

PLATE I.

Figs. 1—4.—Case 1.—Margaret C—. Enlargement of the central canal of the cord.

Figs. 1, 2, which are from the cervical and dorsal regions show the remains of blood in the canal.

Figs. 3, 4, from the lumbar region, represent it as empty. The canal attained in this region the width of one seventh of an inch, or one third the transverse diameter of the cord.

Figs. 5—7.—Case 2.—Mary C—.

Fig. 5 represents an extravasation of blood corpuscles under the arachnoid in the median fissure of the medulla.

Fig. 7 represents a structureless or faintly granular exudation lying between one of the arteries of the corpus striatum and the brain substance.

Fig. 6 shows almost identically the same conditions in the corpus striatum of John P—, Case 5. The artery in this instance is crumpled (this illustration is placed here for comparison with that from the case of Mary C—).

PLATE II.

Fig. 1.—Case 2.—Mary C—.

Perivascular erosion in grey matter of lumbar region, where an artery is seen bordered by a wide space filled with products of nerve disintegration.

Figs. 2, 3.—Case 5.—John P—.

Fig. 2 shows a patch of translucent degeneration, described as sclerosis, in central part of one of the grey horns in the dorsal region. The two sides were affected similarly.

Fig. 3 represents the condition of the white matter of one of the cerebral convolutions. Around each of its arteries is a bulky collection of crystals of hæmatine mixed with the products of nerve degeneration.

Fig. 4.—Case 6.—Louisa W—.

Spots of miliary sclerosis belonging to the *substantia perforata* on the left side—a portion of the base of the brain near the beginning of the Sylvian fissure. An empty artery and a distended vein are seen in the neighbourhood of the spots.

PLATE III.

Fig. 1.—Case 4.—Clara W—.

Mass of extravasated blood in the grey matter of the spinal cord, in the cervical region.

Fig. 2.—Case 7.—Mary O—.

" Pools of exudation," masses of translucent structureless matter in the anterior fissure of the cord, and in the substance of each grey horn—from the cervical region.

Plate 1

Med Chir Trans Vol LiX
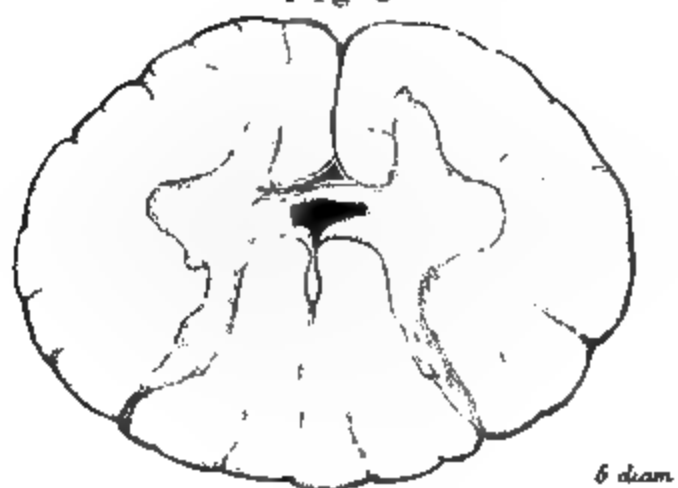
Fig 2

Fig 1

Fig 4

6 diam
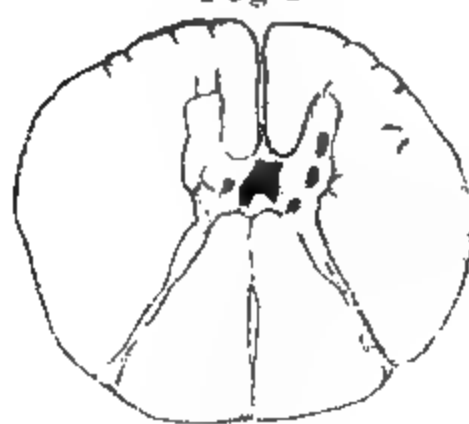
Fig.3

Fig 5

10 diam

Fig 6

Fig 7

100 diam

25 diam

Plate II   Med Chir Trans Vol LIX

ıg 1

10 diam

ıg 3
diam

F ı g 2 *10 diam*

F ı g 4

Plate II.   Med Chir Trans Vol LIX
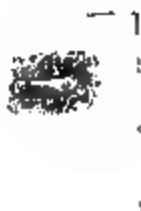
10 diam.

Fig 2

ON THE

USE OF THE ACTUAL CAUTERY

IN THE

ENUCLEATION OF FIBROID TUMOURS OF THE UTERUS.

BY

ROBERT GREENHALGH, M.D.,

PHYSICIAN-ACCOUCHEUR TO, AND LECTURER ON MIDWIFERY AND THE
DISEASES OF WOMEN AND CHILDREN AT, ST. BARTHOLOMEW'S
HOSPITAL; CONSULTING PHYSICIAN TO THE SAMARITAN
HOSPITAL FOR WOMEN; AND CITY OF LONDON
LYING-IN HOSPITAL.

A SOMEWHAT extensive experience of more than twelve years, both in hospital and in private practice, has convinced me of the value of the "Actual Cautery" in many of the local affections of women. Although largely employed on the Continent it has never found favour amongst British practitioners; indeed I have reason to believe few, if any, have had much experience of its use. The cases in which in my hands it has proved especially serviceable are :—

1. In chronic enlargements with induration of the cervix uteri due to inflammatory and fibroid disease.

2. In epithelioma and cancer of the neck of the uterus where the organ is moveable.

3. In some cases of vascular tumour of the meatus urinarius.

4. In slight cases of recto-vaginal and vesico-vaginal fistula.

5. In incontinence of urine due to dilated urethral canal.

6. And in certain cases of interstitial, extra- and intra-uterine fibroid growths.

I purpose to restrict my present remarks to some cases comprised in the two last-named categories, and I believe that the treatment adopted will show some novel features.

CASE 1.—*Interstitial fibroid tumour of the posterior lip and wall of the uterus ; retention of urine and difficult defæcation ; great prostration from severe hæmorrhages ; enucleation by the actual cautery ; recovery.*

M. A— was admitted into St. Bartholomew's Hospital, under my care, during the year 1866, for retention of urine. Æt. 43, married thirteen years, had six children, the last child four years old, with good labours and recoveries, and suckled all her offspring. About three years ago she began to notice a transparent glairy and at times yellow discharge, and about the same period her menses, which hitherto had always recurred about every month, lasting from four to five days, moderate in quantity, and almost painless, gradually became more frequent, persisting from seven to ten days, more profuse and clotted, and usually accompanied with periodic pains like those of labour. During the last six months she had scarcely ever been free from loss of blood, which increased considerably at irregular intervals upon any undue bodily exertion or mental excitement, accompanied at times with discomfort and pain about the lower abdomen, hips and thighs, with gradually increasing difficulty in micturition and defæcation. She was extremely anæmic, with puffiness of the hands, feet, legs, and face ; and of late she had suffered much from palpitation, shortness of breath on the slightest exertion, sense

of faintness with throbbings, and wandering pains in various parts of the body. She appeared free from disease of the heart, lungs, liver, and kidneys, though there was a very audible anæmic bruit, and just a trace of albumen in the urine. She comes of a healthy stock, and until the commencement of her present illness she has enjoyed good health.

The abdomen was somewhat distended, the parietes fat. Occupying the hypogastric region to within an inch of the umbilicus, extending about two inches on each side of the mesial line, was a well-defined elastic swelling, dull on percussion, no fluctuation and no bruit. The vaginal canal was much elongated anteriorly so that it was very difficult to reach the cervix with the finger. The os was found transverse and patulous; its anterior lip lodged immediately below the pubes was found to be nearly obliterated, but the posterior lip could be distinctly traced bulging downwards and backwards, nearly filling up the cavity of the pelvis. The swelling, which extended to within an inch of the vulva, was elastic, immovable and painless on firm pressure. All attempts to raise the tumour out of the pelvis and to pass a sound proved fruitless. The canal of the rectum, containing a large crop of hæmorrhoids, was much flattened by a round elastic swelling, bulging against its anterior wall. An elastic male catheter was introduced with difficulty into the bladder.

There could now be little or no doubt that we had to deal with a large interstitial fleshy fibroid tumour, occupying certainly the lip and probably the posterior wall and fundus of the uterus.

Under the influence of rest in the recumbent posture, a highly nutritious diet, with a moderate amount of stimulants, the administration of the tincture of the sesquichloride of iron in combination with ergot, and great attention to the state of the bowels and bladder, &c., she improved somewhat in aspect and strength, yet the hæmorrhages continued, and the local sufferings increased, leading eventually to so much difficulty in defæcation and

micturition as to render it obvious that the patient must either perish or that something must be done to lessen or to remove the tumour. She was placed upon the operating table, again carefully examined by myself and others, and the previous diagnosis was fully confirmed, but as an eminent physician present expressed an opinion, from a sensation closely resembling fluctuation being imparted to the finger, that the enlarged neck contained fluid, a small trocar was introduced into the most depending part. The withdrawal of the stilette was followed by a jet of arterial blood, but no other fluid escaped. Arterial blood continued to flow pretty freely, jet by jet; and tincture of the perchloride of iron and the nitrate of silver failed to check the hæmorrhage, but it was ultimately arrested by the introduction of an actual cautery, about the size of an ordinary female catheter.

About eight and forty hours afterwards, at my next visit I was astonished to learn that a profuse, watery discharge, slightly tinged with blood had flowed continuously from the vagina, accompanied with pains somewhat resembling after-pains, and my astonishment was increased when I found that I could easily pass my index finger into the opening I had made with the trocar and cautery, and could feel a tumour around which I could pass my finger, and with little difficulty could separate and isolate it from its surrounding attachments. Several days passed without further progress; meanwhile I provided myself with a cautery terminating in a large pointed bulb, which I thought would easily enter the tumour whilst enlarging the opening in its investing capsule. I was gratified to find that within four days the tumour had lost its broad and round form, had become smaller and tapering, and under the influence of the expulsive efforts of the uterus was gradually wedging its way through the dilating opening, until the tumour, being partly extruded through the opening, became firmly impacted and began to slough. Fearing septicæmia I again used the actual cautery and burnt away all the sloughing portion

together with a considerable mass of the growth. Within two days the remainder of the tumour was expelled into the vagina, whence it was easily removed. A month from the commencement of the operative treatment the patient left the hospital without a trace of local disease, and greatly improved in general health.

The tumour was a fleshy fibroid, somewhat larger than the fœtal head at term.

CASE 2.—*Extra-uterine fibroid tumour ; spontaneous slough- ing of the growth ; perforation of the intestine ; perito- nitis ; attempted enucleation by the actual cautery ; death from peritonitis.*

In the autumn of 1866, B. W—, æt. 47, was admitted into the hospital under my care. Had been married twelve years, but was never pregnant. Menstruation, which she thinks commenced about seventeen, recurred at irregular intervals, always deferred, lasting from two to three days, very painful, though since marriage she suffers less and the loss is freer. Has always been sub- ject to a yellow discharge from the vagina. About three years ago she began to lose blood more frequently, every now and then continuously for weeks, sometimes so freely as to amount to severe flooding.

About fourteen months ago the bleeding ceased some- what suddenly, when she was attacked with pains in and about the pelvis, always increased by exertion, and latterly the pains became so severe and constant as to prevent her moving about, and were accompanied with great ten- derness in the abdomen and groins, with considerable constitutional disturbance; the most prominent symptoms being sickness, shiverings, heats of skin, thirst, loss of appetite, sleeplessness, and emaciation.

About seven months ago she began to experience much difficulty at first in micturition, and subsequently in defæcation; but she was relieved from the difficulty in unloading the bowels about three months ago, after

a sudden copious, dark brown, offensive discharge from the bowel, which persisted in varying quantities till her admission.

In addition to the foregoing symptoms, which still persisted in an aggravated form, the abdomen was much distended, tympanitic and very tender to the touch, with an ill-defined sense of resistance in the hypogastrium, and in each iliac fossa.

The vagina anteriorly is much elongated and narrowed, so that the os uteri, round and very small, could with difficulty be reached. The anterior lip is quite oblite-rated. Behind, and apparently continuous with the pos-terior lip, a firm, immovable, slightly tender swelling fills up the whole of the sacral cavity, and extends to within an inch of the vulva. A catheter was passed with difficulty.

The rectum, nearly down to the anus, was so flat-tened by a dense mass pressing against its anterior wall, that it was difficult to introduce the finger, which, when withdrawn, was found to be covered with a melanic-looking and highly offensive fluid.

About this time she was seen and carefully examined by the late Dr. Jeffreson who concurred with my dia-gnosis that her symptoms were wholly due to a large fibroid tumour of the uterus impacted in the pelvis.

In spite of rest, general and local treatment, she con-tinued to get worse.

Bearing in mind the success achieved in my previous case (to which this bore a considerable resemblance), I made an opening with the actual cautery in the most depending part of the growth, into which the instrument entered with astonishing facility.

Two days afterwards, in the presence of Dr. White, of Buffalo, and Dr. Thorowgood, we were surprised to find what appeared to be a very rapid disorganisation of so large a mass, for the finger slid through the opening made by the cautery into a cavity whose boundaries could not be reached by the finger. The poor woman

lingered for a few days, apparently unaffected by the operation, and died with the symptoms of peritonitis.

Post Mortem.—The abdomen was much distended and contained some serum and flakes of lymph, some old, others recent, glueing the intestines together. The pelvic viscera were so firmly matted together by old adhesions that it was with considerable difficulty the several parts could be discriminated. On being removed and carefully examined by Dr. Andrew, the uterus was found high up behind the pubes, to the posterior wall of which was attached, by a slight cellular membrane, a large, thick-walled cyst, with a ragged lining covered by a brown, shreddy, and offensive secretion. Into this cyst three openings were distinctly traced; one communicating with the upper part of the rectum; another with the caecum, *not recent;* and a third at its lower extremity, made by the cautery. It was now obvious that the case was one of *fibroid* tumour of the uterus which had undergone spontaneous disintegration, and in discharging the *débris* through the caecum and rectum had given rise to peritonitis.

Case 3.—*Interstitial fibroid tumour of the posterior lip and wall of the uterus ; enucleation by the actual cautery ; application of écraseur ; recovery.*

On July 16th, 1872, I was requested by Dr. Marriott, of Leicester, to see Mrs. T—, who brought me a note from that gentleman, from which I copy the following particulars. "Mrs. T— has great enlargement of the posterior lip of the os uteri. One and a half years ago I was called in consultation and performed craniotomy when we had the greatest difficulty for three or four hours in completing the delivery. Seven months ago I induced premature labour, the labour was most satisfactory, all being over in two or three hours, but the child only lived a short time. Some time ago the posterior lip was ulcerated and enlarged, but during pregnancy it enlarged

enormously, so as to block up the pelvic cavity to a great degree. I treated her with bromide of potassium, and locally with solutions of nitrate of silver, chloride of zinc, &c., and the ulcer healed, but directly pregnancy took place it began to enlarge as badly as ever." She told me that she was thirty-eight years of age, had been married thirteen years, had borne eight living children and undergone four miscarriages. With the exception of an occasional sensation of weight and bearing-down of the womb, for which she had been wearing a support, slight pain over the left hip at times, and a watery and mattery discharge, her health had been good and seemed so still. Her two last catamenial periods had been somewhat freer, but otherwise normal.

Examination.—Abdomen normal, parietes fat, vagina capacious and lax. Uterus lying within about an inch of the vulva, posterior lip and body considerably enlarged, somewhat elastic to the touch, anterior lip thin and healthy, drawn in a crescentic shape over the posterior lip. Sound entered the .uterus four inches with ease in the normal direction. The speculum revealed the posterior lip red and abraded. For the next three months, notwithstanding repeated puncturings and the application of iodised cotton and glycerine pledgets to the cervix, with other means, the enlargement continued to increase. On the 17th of November I proposed, and Dr. Marriott readily consented to the enucleation of the growth by the actual cautery. As Mrs. T— was in full health, suffering little inconvenience from her local affection, I deemed it expedient to seek the opinions of Mr. Spencer Wells, Dr. Meadows, and Dr. Desmond, an old friend of the family, who all concurred as to the nature and situation of the growth, and feasibility of the plan recommended for its removal. Accordingly, on the 6th of December, the patient being under the influence of chloroform, I applied an olive-shaped actual cautery to the most accessible part of the lip, posterior to the os uteri, and made an opening sufficiently large to admit with ease my index

finger, when I came upon the tumour and found that I could easily break down the cellular attachments of its investing capsule, which was soft and about an inch in thickness. During the following two days a considerable amount of serous fluid tinged with blood continued to ooze through the openings. The investing capsule having become as thin as parchment, the tumour tending to descend, and the orifice dilating, we made daily attempts with the finger to free the growth from its attachments.

At this stage, I was obliged to leave England, and Dr. Meadows on December 16th kindly took charge of my patient. On my return, after an absence of four days, I learnt that under the expulsive efforts of the uterus a considerable portion of the growth, about the size of a large foetal head, had become impacted in the opening, and, commencing to slough, was judiciously removed by Dr. Meadows with the wire écraseur. The remainder I detached with the finger, and shortly afterwards extracted, Dr. Meadows being present. Its removal was followed in about two hours by rather free hæmorrhage, which was speedily arrested by plugging with cotton wool saturated with tincture of the perchloride of iron.

With the exception of vomiting due to the chloroform, and some trifling general and local disturbance, the patient made an excellent recovery, and now, more than two years after the operation, is in excellent health, and free from any evidence of a return of the growth.

Mrs. T— again became pregnant, and having passed through a normal child-bearing was at term on September 28th, 1875, about two years and three quarters after the operation, delivered by Dr. Marriott of a fine, fat, full-grown, male child, after an easy labour of between three and four hours' duration. She made a satisfactory recovery. No trace of a tumour could be detected during pregnancy, at parturition, nor subsequently.

CASE 4.—*Intra-uterine fibroid tumour of the uterus; severe and frequent hæmorrhages; alarming prostration; removal by the actual cautery; application of the écraseur; recovery.*

On June 4th, 1873, C. S— was admitted into St. Bartholomew's Hospital under my care. Æt. 47. Married many years. Never pregnant. Stated that about sixteen months ago, her periods, which hitherto had always been normal, began to be excessive in quantity, accompanied by forcing and dragging pains about the pelvis, constipation, painful and difficult micturition, amounting, on three occasions, to retention of urine. Although she had been under treatment and lying up she had derived but little benefit. On her admission she was in a very prostrate state, flooding profusely, able to retain scarcely anything on her stomach, sleepless, and suffering intense headache.

Examination.—Abdomen greatly distended and tympanitic, somewhat tender on pressure. A well-defined, round, firm swelling occupied the hypogastric region, extending to within an inch and a half of the umbilicus, and to a like distance on each side of the mesial line.

A large, round, very dense mass, smooth, except in one part, which was rough and about the size of a shilling, occupied the roof of the short vagina. This proved to be the os uteri, whose circumference, including the lower segment of the neck, was tightly stretched over the growth. The finger could be passed around the growth, except at the anterior part of the cervix uteri, to which it was firmly adherent.

The hæmorrhages having been arrested by repeated pluggings, and her general condition improved by light nourishment, and reduced iron with pepsine, on July 2nd a cautious attempt was made with the finger to break down the numerous bands which bound the tumour to the uterus, the os becoming dilated by the gradual extrusion of the growth. On the 31st, having much improved in every respect, she was placed on the operating table,

and an olive-shaped actual cautery was passed, through a wooden speculum, freely up the centre of the growth. The operation was painless and no bad symptoms followed.

On August 7th, the tumour had wedged itself through a more widely dilated os, into which the finger could be passed some distance. The cautery was again applied and effected the destruction of a large portion of the tumour. Slight febrile disturbance followed, with abdominal pain, foetid vaginal discharge, and the escape of sloughing tissue, which symptoms were relieved by the frequent injection of Condy's fluid and water.

On the 14th and 21st the cautery was repeated, followed by daily attempts with the finger to free the tumour from its attachments. This brought on periodic forcing pain, and the ultimate expulsion of a considerable mass.

She continued to mend well until the 1st of September, when she was seized with cold chills succeeded by profuse perspiration, quick pulse, red patchy tongue, and considerable abdominal pain, with thick, yellow vaginal discharge.

On the 11th, her general condition having improved, traction was made upon the tumour with the vulsellum forceps, and the cautery applied for the fourth time.

On the 18th large portions of the extruded mass were cut away.

On the 26th and October 2nd, attempts were made to break up the mass by the insertion of scissors into the centre of the tumour, which was found to be extremely dense. No haemorrhage followed.

At this juncture I started for Egypt, when she was placed under the care of Dr. Hope, to whom I am indebted for the following details.

Between October 2nd and November 6th, she suffered much in general health, and the discharge from the vagina was most offensive. Several attempts with but partial success were made to disintegrate and detach the tumour. On the latter date, the tumour being within easier reach,

first one and then another strong écraseur wire was passed as high as possible over the growth, but it proved to be so dense that both wires broke. At that time no further attempts to remove the growth were made; but meanwhile the vagina was frequently syringed out with carbolic acid and water, and quinine and ergot were administered. At the end of three days the lower portion of the tumour, which had become strangulated by the écraseur, separated, and was removed from the vagina.

On the 27th of November and 18th of December considerable portions of the tumour having descended were removed by the écraseur.

From this date to January 15th, 1874, her condition varied, but on the whole she gained ground. Another large portion of the tumour was removed by the écraseur.

Feb. 2nd.—Patient better. The remainder of the growth of large size but narrower and less expanded than the previous pieces, attached by a somewhat broad base, was now separated from its attachments to the uterus by a curved curette, and subsequently removed by the écraseur.

From this time the general condition and aspect greatly improved, the uterus rapidly decreased in volume, and the patient, at her own request, left the Hospital on the 7th of February, apparently quite convalescent.

CASE 5.—*Interstitial fibroid tumour of the anterior lip and wall of the uterus ; severe and frequent hæmorrhages ; alarming prostration; removal by the actual cautery, écraseur, and hand ; death from embolism six days after the removal of tumour.*

A. J— was admitted into St. Bartholomew's Hospital under my care, on August 11th, 1874. Æt. 30. Married seven years, never pregnant. The catamenia commenced at fifteen, continued normal till the previous December, when they became freer, lasting seven days.

In January last, shortly after a period, she was seized

with a severe flooding. Since then she has been subject
at varying intervals to frequent large losses of blood, unat-
tended with pain. She was very anæmic and much emaci-
ated; pulse quick and feeble; lips, gums, and tongue
very pale; scarcely any appetite, and slept badly. As she
was losing blood freely with coagula, she was ordered
the tincture of Indian hemp and ergot, with generous
diet.

On the 20th, bleeding having ceased, careful examina-
tion revealed a firm, somewhat round, uniform swelling,
dull on percussion, and slightly moveable, occupying the
hypogastric, iliac, and umbilical regions, extending upwards
to within two inches of the ensiform cartilage.

External orifice of vagina very small, otherwise normal.

Uterus low in the pelvis. Os slightly patulous; anterior
lip occupied by a firm, round, uniform swelling, which
extended to within about an inch and a half of the vulva,
and upwards, anteriorly, as far as the finger could reach,
bulging into the cervix, and stretching the posterior and
attenuated lip into a crescentic form. Ready impulse was
communicated to the os by pressure upon the abdominal
tumour. The sound, with its concavity forwards, entered
the uterine cavity posterior to the uterine tumour four
inches with ease. Free bleeding followed its use.

Sept. 3rd.—Had been steadily improving, and had not
lost blood for a week.

10th.—She was put under the influence of chloroform,
and then with an oval-shaped actual cautery, a transverse
opening was made sufficiently large to admit the index finger
in the most accessible part of the enlarged lip, anterior to
the os uteri. Through this opening the tumour could be
distinctly felt, and its cellular attachments broken up. A
free, serous discharge, slightly tinged with blood, followed
this operation, but no untoward symptoms, and her health
still continued to improve.

On September 21st the growth was found pressing
firmly against the opening, and an olive-shaped cautery
was freely passed into its centre and lower segment

with the view of lessening the size of the tumour and rendering it more conical.

29th.—A considerable portion of the centre of the tumour was found protruding and was again destroyed by the cautery. The operation was followed by occasional attacks of expulsive pains with vomiting and further descents of the growth into the vagina.

Oct. 8th.—Improving satisfactorily, feels occasional forcing pains ; abdominal swelling much decreased.

22nd.—General condition much improved. A large mass protruding through the opening was removed with the écraseur wire. Much force was required, the structure of the growth being very dense with coarse fibroid intersections.

Nov. 5th, 12th, 19th and 26th.—Large masses were removed with the écraseur, and from time to time numerous bands of attachment were broken up by the finger. Abdominal tumour much decreased. Ordered firm pressure by abdominal belt, and ergot. Patient doing well.

Dec. 7th.—Removed a large portion of the growth, less dense in structure.

17th.—Had suffered the last few days from vomiting, hot skin, rapid pulse and sanious vaginal discharge. Large mass removed.

21st.—Vomiting and fever increased ; vaginal discharge very offensive. Ordered salines with hydrocyanic acid, ice, champagne, and frequent syringing with Condy's fluid and water.

25th.—Some pain and tenderness in the popliteal space of left leg with swelling about the ankle ; no redness nor hardness along the course of the veins ; took food fairly well. Ordered ferruginous tonic.

31st.—Removed a large mass protruding into the vagina. This gave exit to some pent-up offensive discharge.

Jan. 11th, 1875.—Much improved since last operation, being able to get up each evening. Within the opening an irregular, much fissured mass could be felt, and its extent

now fairly defined.   Ordered ergot, and pressure by abdominal belt.

18th.—Some portions of the tumour were removed by the scissors; this was followed by free bleeding, requiring the plug.

21st.—Ordered india rubber bags to be introduced daily into the vagina and gradually inflated so as to dilate that canal, but as this procedure induced considerable irritation it was discontinued.

27th.—In the last few days severe abdominal pain set in with distressing vomiting, rapid pulse, hot skin and very fœtid vaginal discharge, with much abdominal distension, especially in the left iliac region.   A most offensive sloughing mass occupied the vagina and extended continuous with the tumour within the uterus.   The portion of the mass within reach was removed under æther when the abdominal pain ceased, though the other symptoms, with restlessness, great anxiety and facial sallowness, persisted.   Vagina to be frequently syringed out with Condy's fluid and water; beef juice, brandy and opium to be injected into the rectum every fourth hour.

28th.—Again put under æther when the hand, with great difficulty, was introduced into the vagina, and a large highly offensive slough was removed.

29th.—General condition somewhat improved; freedom from pain; vomiting still distressing; discharge less fœtid. Nutritive enemata continued.

30th.—General condition very distressing; sickness almost incessant; discharge from vagina highly offensive. Again put under æther when the hand was introduced into the opening in the uterus and a large putrid mass was removed with much difficulty owing to the vigorous contractions of the uterus, a small dense portion being so firmly attached to the anterior wall that it could not be separated.

31st.—Had passed a comfortable night, pulse fairly good; tongue clean and moist, temperature normal, abdomen flat, no tenderness on pressure, no trace of tumour to

.be detected above the brim of pelvis ; still unable to take nourishment by the mouth.   Nutritive enemata continued.

Feb. 1st.—General condition improved, slept well, able to retain some nourishment on the stomach; tongue moist, slightly furred; vagina very tender; opening in the uterus contracted to about the size of a florin ; within the opening a small firm portion of the tumour was detected; discharge slight and only faintly fœtid.   During the two days following she continued to gain ground when suddenly she was seized with severe dyspnœa again and again recurring, followed on the fifth attack by alarming pros tration, from which she never rallied.   She remained sensible to within an hour of her death, when coma super vened and she expired apparently from embolism, on the evening of the 5th of February, six days after the removal of the tumour.   No post-mortem was permitted.

The tumour was about the size of an adult head and a half, and weighed seven pounds, one ounce.

Thus, of the five cases, all to be regarded as excep tional, three, Nos. 1, 3 and 5, were intra-mural; one, No. 2, extra-mural, and one, No. 4 intra-uterine.   Three cases, Nos. 1, 3 and 4 recovered; two, Nos. 2 and 5 died; one, No. 2, from peritonitis due to spontaneous sloughing of the tumour and perforation of the intestine prior to any surgical interference; the other, No. 5, from embolism six days after the removal of the tumour.   Both these fatal cases, as well as Nos. 1 and 4, were in a desperate condition from losses of blood previous to any operative procedures.

Although I agree with the generally expressed opinion that the large majority of cases of diffused fibroid deposits and fibroid outgrowths of the uterus are but slightly, if at all, amenable to treatment, yet I am convinced that in the early stage a certain number of fibroids, which rarely come under our charge, can be arrested and even removed by conjoined general and local means. Putting aside this question, regarding which I have reason

to believe there would be found great diversity of opinion, it is on all hands admitted that every now and then cases do occur which, either from the severe hæmorrhages they occasion or from the serious mechanical impediment they produce, do imperatively demand surgical interference. The cases just recorded appear to me to afford typical examples of this necessity. Enucleation in many instances has been practised with very successful results. The usual means adopted are either the free dilatation of the cervix by tents or its division by the metrotome, the subsequent incision into the capsule being followed by the detachment and removal of the growth. It has fallen to my lot to witness more than one case of dangerous and even fatal results through the irritation induced by the process of dilatation, and severe hæmorrhage and septicæmia from the cutting procedure. Hence, it appears to me that some method calculated to obviate such casualties was much to be desired. The use of the actual cautery to arrest the bleeding and the encouraging results which followed in Case No. 1, led me to hope that in this agent I had found safe and sure means to accomplish my purpose; and experience has ratified this anticipation. I will now specify those points, wherein I consider that the cautery possesses advantages over every method hitherto suggested for the treatment of uterine tumours.

1. The facility of application.
2. It occasions but little pain.
3. The rapidity of its action.
4. It occasions no bleeding.
5. No plugging is needed.
6. The charred opening is not favorable to absorption.
7. No offensive discharge from the charred tissue.
8. The opening is readily dilatable without inducing bleeding.
9. Free manipulation is practicable through the opening immediately after cauterisation.
10. It does no damage to the lining membrane of the uterus,

11. Portions of the tumour may be rapidly destroyed, its size reduced and its lower segment rendered conical, thereby facilitating dilatation of the opening and the subsequent detachment, expulsion, or extraction of the morbid growth.

It is worthy of remark that spontaneous expulsive efforts shortly followed the use of the cautery, it seeming more or less to reduce the density of the tumours. In conclusion I would specially invite attention to three points in connection with the enucleation of fibroid tumours of the uterus :

I. The advisability of the gradual detachment of the morbid growth from its investing capsule, especially when the tumour is large or when the patient has been much reduced by previous hæmorrhages. The observance of this precaution will tend to prevent further losses of blood and to secure more perfect contraction of the surrounding tissues ; moreover the chance of offensive discharge being retained is thus almost certainly obviated.

II. The removal at each operation of that portion only of the tumour which bulges external to the opening, so that the latter is kept dilated and the chance of its closure upon the remainder of the growth is avoided.

III. The speedy destruction by the cautery or removal by the écraseur or hand, should sloughing of the tumour ensue.

Although I freely admit that it would be premature to generalise upon the foregoing five cases, and to found thereon any definite plan of treatment, still the results are, I think, sufficiently encouraging to warrant further trials of the actual cautery in certain exceptional and grave cases of fibroid tumours of the uterus.

ON

AORTIC ANEURISM IN THE ARMY,

AND THE CONDITIONS ASSOCIATED WITH IT.

BY

FRANCIS H. WELCH, F.R.C.S.,

SURGEON, ARMY MEDICAL DEPARTMENT, AND ASSISTANT PROFESSOR OF
PATHOLOGY, ARMY MEDICAL SCHOOL, NETLEY.

(COMMUNICATED BY GEO. D. POLLOCK, F.R.C.S.)

To any one perusing the literature of this subject in
the text books of our schools, the idea must occur how
unstable and various are the opinions advanced in regard
to the causation and surroundings of these vascular lesions,
and how little knowledge we possess of the disease which
is placed upon a basis not open to question, and beyond
the region of doubt. There is a strange discrepancy of
opinion current in the military and civil segments of the
medical community both in reference to ascribed external
physical causations of the lesion, as well as to internal
generating conditions; among the latter I especially
include the influence of the syphilitic virus, an opinion
firmly held by many military medical men, but more than
doubted by the civil medical community at large, the

reason probably being that (in the words of the late Professor Parkes) " no analysis of cases has been made, and therefore at present its effects must be considered uncertain."

Doubtless it may be said by some that the circumstances attending military life are so special and peculiar as to make the deductions arrived at, in reference to any disease prevalent among soldiers, comparatively worthless for application to the community at large; and although this may hold good of certain limited spheres of medical and surgical science, yet I think it will be patent to all that such an objection has no validity in regard to the subject under discussion in this paper. Human nature is the same, to be impressed by general agencies, whether clothed by the uniform or under the diversified garment of civil occupation, and it may moreover be urged that the surrounding conditions and circumstances of a soldier's life are so precise and well known as compared with those of his civil brother as to render deductions on disease made from this segment of the community of much greater value and trustworthiness.

Thirty-four fatal cases of aortic aneurism are fully detailed in the pathological records of the Royal Victoria Hospital, Netley, and these form the basis of the paper. The average death age is 32 years but ranging from 26 to 42 with an average period of service of 12 years, and ranging from 4 to $21\frac{1}{4}$ years, thus embracing the entire course of military life. The average duration of the lesion is $1\frac{1}{13}$ years, varying from $3\frac{1}{2}$ months to $2\frac{8}{13}$ years, but this is necessarily calculated from the time the disease became sufficiently pronounced to render the sufferer cognisant of its existence, to the date of death, and consequently is decidedly within the period from which the dilatation of vessel dates; it represents what may be termed the clinical duration of the malady. In 5 cases the aortic dilatation was multiple; in one two sacs projected from the transverse portion of the arch, one superiorly, one inferiorly; in one with two sacs from the transverse arch

posteriorly, was a third (largest and death-causing) from the descending thoracic ; in two there was a thoracic and abdominal sac ; and in one, with a thoracic sac were three abdominal sacs ; and in two instances an aneurism of the innominate artery was conjoined with the aortic dilatation. As regards the form of the dilatation in 6 this was fusiform, viz. 5 thoracic (3 embracing ascending and transverse portion of arch, 1 transverse, 1 transverse and descending portion), and 1 abdominal; in the remainder the sacculated variety was found. In 1, the sacculated aneurism came under the category of false, the sac seated in the heart's substance and taking origin from immediately above the aortic valve ; in 4 of the fusiform cases there was a false sac in connection with the original sac ; in one of the sacculated cases this also existed ; and in one a dissecting aneurism was situated in the walls of a sacculated abdominal aneurism. On the point of site of the sac the following table is explanatory, brought out as a percentage to exemplify relative frequency.

Ascending portion of arch . . .	26·1 per cent.	Arch of aorta 64 per cent.
Ascending and transverse combined [1] .	9·3 „	
Transverse	26·1 „	
Transverse and descending combined [1] .	2·3 „	
Descending thoracic aorta . . .	16·6 „	
Abdominal aorta	19· „	; the thoracic

lesion predominating over the abdominal a little in excess of the ratio of 5 to 1.

In 24 of the cases the condition of the heart was clearly ascertained as follows :—in 9 or 37·5 per cent. it was normal ; in 11 or 45·8 per cent. (taking 10·07 oz. as the average weight of the viscus in health), it was enlarged, in 6 of the cases generally, in 5 limited to left ventricular hypertrophy ; in 3 or 12·5 per cent. it was atrophied, reduced in size, but normal in structure ; in 1 or 4·1 per cent. it was fattily degenerated. Hence these cases tend to show that a heart diverging from the normal standard in size or condition is no necessary asso-

[1] i. e., both portions implicated in the sac.

ciate of aortic aneurism, and they also indicate that the
cause of aortic aneurism cannot as a rule be linked with
an over-acting viscus extra-forcibly ejecting the blood
and overcoming the normal recoil of the arterial walls.
The inference is rather that divergence in form and
structure of the heart follows the arterial lesion, in pro-
portion to the obstacle to the blood current and to the
constitutional capacity of the system to meet the altered
demands of the viscus.

Turning to the statistics of its dispersion throughout
the service, we find that during the decennial period,
1863—1872, the loss by combined deaths and invaliding
from aortic aneurisms alone was as follows in the three
chief countries now occupied by British troops:

Home force (cavalry, artillery, and infantry) . ·55 per 1000 of strength.
Mediterranean garrisons (infantry, artillery,
      and engineers) . . . . . . ·50   „   „
India (cavalry, artillery, infantry, engineers, &c.) ·47   „   „

The home segment, with an average yearly strength of
68,760 men, during the period of ten years suffered an
average annual loss from aortic aneurisms of thirty-eight
men.

The component branches of the service constituting
the home segment during this period gave a loss as
under.

Cavalry (household and line) . . . . ·53 per 1000 of strength.
Artillery . . . . ·69   „   „
Infantry (foot guards and line regiments) . . ·52   „   „

Inspector General Lawson, taking the station of Alder-
shot as an exponent, concluded from the statistics of the
disease during 1867–8, "that aneurism was not con-
nected with any particular arm of the service, and even
in the infantry was very irregularly distributed," for
example, "out of an average of 10·2 foot corps, deaths
from aneurism appeared in four of them only, while one
third of the whole number of the cases of the disease was

met with in one regiment, and all apparently under the same conditions of dress, duties, &c." ('Blue Book' for 1868, p. 269.)

These figures indicate that the causes of aortic aneurism in the service are generally dispersed, not peculiar to climate, station, segment, or branch, while it is equally apparent that in the components of the respective branches of the service considerable diversity exists, and the thirty-four cases of this paper show that these causes are not connected with any special age, nor any condition of system brought about by mere length of service. What these causes are is an important problem to solve, one that it is impossible to over-estimate; and on this point the evidence furnished by the morbid anatomy and life-history of these cases seems conclusive. The deductions arrived at may be placed in the form of two propositions.

1. *That the aneurismal tumours are associated with, and preceded by, a diseased condition of the contiguous layers of the internal and middle coats of the vessel—a tissue growth terminating in degeneration—which, by impairing the elasticity and contractility of the walls, allows of their expansion and dilatation under the tension of normal arterial blood pressure, or this abnormally increased by any cause.*

This diseased state of the vessel walls comes under the nomenclature of atheroma, an extremely unsatisfactory designation, inasmuch as, taking the word in its present accepted meaning as expressive of a phase of fatty degeneration, it conveys but part of a truth, and that not the most important, and allows of the accumulation under one heading of structural changes divergent in origin and progress.

This disease in its early stage is met with as small, elevated, translucent dots, situated immediately beneath the serous surface, irregularly scattered or linearly arranged, by coalescence assuming any shape of outline; they are due to material added.[1] They increase in thick-

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[1] I find that the combined internal and middle coats of the aorta in health average $\frac{4\cdot5}{100}$ of an inch in thickness, while in this disease I have observed a thickness of $\frac{8}{100}$ inch, due to excess of bulk of the internal coat alone from material added to it.

ness and extent, and subsequently become opaque white, or occasionally mottled red or black from blood-colouring, and from their origin in scattered foci of tissue germination, by coalescence, the inner surface of the vessel is rendered nodulated and furrowed, the furrows not uncommonly being linear and in the direction of the vessel. A vertical section through the diseased walls shows the added material as hillocks of firm tissue between the internal and middle coats, and by dissection this tissue is found mainly to be connected with the internal one (Pl. IV, figs. 1 and 2).

Under the microscope the new tissue consists of fibrous tissue cells and fibres, with very numerous nuclei in a free state (Pl. IV, fig. 3), apparently interspersed among the normal constituents; it comes under the category of growths due to localised germination of normal tissue elements, a phase of the so-called chronic inflammation, an end-arteritis. Up to this point the calibre of the vessel is unquestionably encroached upon; instead of the area being increased it is decreased from the projection within it of the more or less numerous nodular elevations (Pl. IV, fig. 1). Should the disease rest here, the vessel walls become permanently thickened, indurated, with a loss of elasticity; but as a rule the new material, following the general law of abnormal tissues, retrogresses, the node breaks up from the centre, fat globules, caseous-like particles, phosphatic crystals, and cholesterine gradually replace the fibrous tissue elements (Pl. IV, fig 6). This phase is, to the naked eye, associated with increasing opacity and softening of the patch, and then rightly comes within the strict meaning of atheroma—a reduction to a gruel-like fluid; the elevation of the inner surface of the vessel disappears, the coats reach their normal thickness or become thinner, the internal one still retaining its glistening appearance, but thrown into wrinkles by the gradually absorbed subjacent node; and so the aorta may be left with a cicatricial-like puckering of its walls and internal roughness, but no dilatation. But as a rule the formation

of abnormal material does not proceed to any great degree without implication of the inner portion of the middle coat, and consequent on the pressure from the added material and the degradation ensuing in it the walls of the vessel corresponding to the patch become decidedly impaired in structure and function. They lose their property of contracting and recoiling after distension by the blood current, the distension remains and is gradually increased, ultimately reaching a degree which brings it within the category of pronounced aneurism.

As above said the serous surface of the vessel undergoes but little if any change, retaining its translucency and glistening aspect, thrown only into folds and rugæ by the changes ensuing in the subjacent node, but occasionally it is seen to be implicated, and this in three ways: —(a) By the formation on its free surface of a delicate lymphy layer, generally stained by the colouring matter of the blood, and made up of extremely delicate interlacing fibres and nuclei. (b) By the transformation of its substance into a smooth, glistening, thin, friable, inorganic stratum, answering to the so-called ossification of arteries, and occupying the free surface of the vessel, lying on a subjacent fibroid patch, or one in process of softening. This may occasionally be seen forming a ring at the commencement of the aorta for about three quarters of an inch in extent, above which, in the continuity of the vessel, will be nodes firm or softening, gradually merging into healthy texture beyond. (c) It may be implicated in the disease-process, and gradually disintegrate, forming, with the subjacent changes, a sharp cut ulcer with walls and base studded with particles of degenerate tissue (Pl. IV, fig. 5). The middle coat is also occasionally seen to be involved by the production within its fibres of a distinctly circumscribed fibrous tissue nodule answering to the node formed in the voluntary muscles, and undergoing the same phases as the sub-serous thickening (Pl. IV, fig. 4, d). Also in the external coat may be noted the occasional presence within its loose meshes of

small, circumscribed, microscopic nodules of nuclear adenoid tissue,—lymphatic outgrowths, miliary tubercle; these being connected with each other by cordlike processes of similar material, and also with similar processes passing through the middle coat to a disintegrating internal patch. These examples suggest the normal distribution of the lymphatic tissue in the large vessels, and also outgrowths from the same under infection from the degenerating nodule towards the inside of the vessel, similar to the phases observed elsewhere under inoculation and artificial tuberculosis. Also occasionally, before any dilatation of the vessel wall has ensued, corresponding to the internal patch, is augmented vascularity of the external coat, being apparently the first stage in the natural process which ultimately forms the aneurismal sac; an intermediate stage—that of tissue production and thickening—is seen in Pl. IV, fig. 1 d.

As before mentioned the disease commences in separate foci ultimately more or less coalescing, and the condition of the nodules found in any given case leads to the inference either that these foci succeed each other as successive crops at different intervals or that the changes ensuing in the several masses of abnormal material are not at all uniform in time or degree. For example, in the same vessel we may find the following : (1) pouching or dilatation, general or localised, with nodulated and corrugated walls,—the most advanced of the disease already gone on to immature aneurism ; (2) cicatricial-like puckering of the inner surface, with walls either of normal thickness or slightly thinned, but with no dilatation— nodes completely retrogressed and absorbed ; (3) patches of thickening, opaque, soft, and friable—retrogressing ; (4) nodes firm and semi-translucent, encroaching on the area of the vessel. Under these conditions it is clear either that the nodes are not all formed at the same time, or that if so formed there are great divergencies in time in the ulterior changes ; the former, however, would appear to be the explanation. There is also marked

variation in the extent of vessel implicated—sometimes there is one circumscribed patch, oval in outline, and this is more often seen in the abdominal aorta; more generally the patch is irregular, more or less encircling the calibre of the vessel; at the commencement of the aorta the predominating forms are either a distinct ring, or a localised patch in one or more of the sinuses of Valsalva—under the latter phase very intense in degree generally; not uncommonly, from the site of commencement, characterised by the greater intensity and most advanced stage, the disease is seen to radiate even throughout the entire aorta, the extreme limits being marked by outlying small isolated nodules. As regards regional selection, the commencement of the aorta is at the head of the list, then the transverse portion of arch or abdominal portion just beyond the diaphragm ; and in either of these or other site selected the disease may be found localised with a complete freedom of any other part of the aortic continuity.

I have dwelt thus long on the vascular disease because it seems to me to be the key to all the ulterior changes, and because a knowledge of its phases and their modifications appears to explain much that is obscure and questioned in the natural history of the aneurismal tumour. It is clear that the extent and subsequent phase of the node determine the ulterior results, and the nature and kind of the aneurismal lesion. So long as the added fibroid material remains as such, or should it be limited in extent and on retrogression be absorbed without impairment of the function of the walls, no dilatation will ensue. But should the natural function of the walls be impaired, then in the event of the disease being generally dispersed over the whole calibre and uniform in degree of degradation, we get a general dilatation, a fusiform aneurism ; should the lesion be limited in extent, a mere patch, the dilatation will be limited and the aneurism sacculated, and this is not uncommonly seen combined with the former form from one patch of disease

out of a mass generally dispersed being in advance of the rest, and so inducing a sacculated aneurism projecting from a fusiform kind; should the internal coat of the vessel be implicated in the degeneration forming an open ulcer, then all the elements for the dissecting aneurism are present. It will also be apparent that an aneurism may arise from a local patch of disease, and yet no disease may be found elsewhere in the vessel; a feature clearly illustrated in the natural history of aortic nodes, and which I believe forms often the true explanation of many of those so-called examples of "aneurisms not preceded by atheromatous change," the deduction being generally made from the absence of disease elsewhere in the vessel, and the fact overlooked that the stages of thickening and atheroma are necessarily past and gone before the aneurism can ensue.

The evidence furnished by the post-mortem records of this hospital clearly shows that it is impossible in pathology to separate the aortic disease from the aneurismal lesion, the former being the precursor of the latter, and this is seen not only in those examples coming under the category of aneurism, and classified as such, but also in those cases of disease elucidated post mortem, the cause of death being otherwise than arterial lesion, but in which the aortic disease is present yet not sufficiently advanced or pronounced to give it a maximum importance in the determination of death. From the fibrous node in the internal and middle coat to the aneurism is a connected chain, which commencing as a tissue growth, abnormal in origin, leads through a fatty and caseous degeneration of the formed material to impairment of the resiliency of the arterial walls, and so under internal blood pressure to dilatation. That this degeneration of the vessel coats is no mere result of age-changes, is clear from the death ages of these cases; that a tissue growth precedes the degeneration is unquestionable; hence the important point in ætiology is to find out the causation of the growth—the conditions under which this germination of the con-

tiguous layers of the internal and middle coats of the vessel originates; for the subsequent degeneration to which it is liable, and which lays the foundation for the aneurismal dilatation, is a phase common to most abnormal growths and some normal tissues under deteriorated states of the system.

But there is another condition of the aortic walls also included under atheroma. It is met with as a more or less diffused opacity seen from the inner surface of the vessel, in the form of an irregular patch or streak; its seat being in the internal or possibly the inner layer of the middle coat. But there is no thickening of tissue conjoined with it, and no added material, and in all the examples I can find in these records it is never seen otherwise than as an opacity with no anterior or succeeding phase; I have not been able to connect it with any dilatation of the vessel, it appears a mere passive condition. Microscopically it is sometimes unquestionably fatty degeneration, and apparently of normal tissue; sometimes no fatty change can be detected, and the cause of the opalescence is far from clear. In its extent, degree, structure and ulterior results, it diverges from the nodular growth; it fairly comes under the definition of a limited opacity of the internal arterial wall, and does not appear to be followed by any deleterious result within the soldier's service—eighteen to forty years of age—and under the conditions of military life.

Thus, these two forms of aortic disease are included under atheroma : the one a passive degenerative phase apparently innocuous, the other a fibrous tissue growth with sequelæ as follow:—(a), it may encroach on the calibre of the aorta and produce a permanent curtailment of its area; (b), it may induce an indurated and inelastic condition of the walls antagonistic to the normal expansion and recoil under the blood current; (c), when seated in the ascending portion of the vessel it may so obstruct the onward passage of the blood as to lead to hypertrophy or dilatation of the left ventricle and death through the

damming back of the venous blood current; (d), by extension to the aortic valve it may effect the same end; (e), by softening and impairment of function of the walls it may illustrate aneurismal lesion, and (f), by its roughened surface, it may lead to fibrinous deposition, and through this to embolic transference to distant parts.[1]

(2nd Proposition). *That these two forms of textural derangement of the aorta are dissimilar in origin and causation; that the limited passive opacity is connected with long-standing diseases of various kinds inducing a diminished vitality of the system at large; that the structural growth is in the major number of instances associated with syphilis, and in a minor degree with rheumatism and alcoholism, as causations: hence it follows that, as the latter phase is the commencement of that pathological sequence of events under one aspect terminating as aneurism, the means for the prevention of the aneurismal tumour must be essentially directed towards the elimination of the special exciting agencies.*

Taking in the first place the thirty-four cases of aneurism, the matter stands thus :

(a) In constitutions undoubtedly syphilitic and nothing otherwise, 17 or 50 per cent.; (b) in constitutions probably syphilitic, but not beyond doubt, 5 or 14·7 per cent.; (c) with an acute rheumatic diathesis, 2 or 5·8 per cent.; (d) with excessive intemperance, but no other disease, 2 or 5·8 per cent.; (e) with syphilitic infection, but also rheumatism and alcoholism, 1 or 2·94 per cent.; (f) of no known condition of system from absence of reports, 6 or 17·64 per cent.; (g) with history, but no ascertainable condition of system, 1 or 2·94 per cent.

This analysis is based upon the "medical history

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[1] Equally also in the vessels of the brain, both large and small, we see similar changes of thickening, dilatation, blood obstruction, thrombosis and embolism, leading to impaired and irregular function of the nerve centres, softening, and death, and under conditions of system similar to the aortic disease. The records of this hospital illustrate such cases, and indicate them as *one* form of brain disease due to the syphilitic virus.

sheet " of the man, detailing his diseases from the date of entry into the service, and the post-mortem facts. One or two of the headings require explanation. By "probably syphilitic but not beyond doubt" is meant, for example, that with a history of primary sore there are conjoined post-mortem lesions whose import might possibly be questioned, such as induration and ulceration of tonsils, or that with no history of primary sore the post-mortem lesions, although strongly suggestive of syphilis, cannot, without doubt, be classed as such ; yet in these examples it must be remembered that there were no other diseased conditions with which to connect the lesions. Under (*f*) are embraced those cases whose life-records are not forthcoming, and in which the post-mortem data throw no conclusive light upon associated systemic conditions.

Hence, it is clear that 50 per cent. *at least* of these aortic aneurisms occurred in subjects with syphilitic infection, and with no other ascertainable conditions present to neutralise the deductions arrived at on the point of causation ; while, on the other hand, the only other recognisable conditions present with which to connect these lesions were, the acute rheumatic diathesis and alcoholism, each represented by a percentage of 5·8.[1]

But as the aneurism is only one sequel of aortic nodulation, it is very essential, on the point of aetiology, not only to regard the surroundings of one of the pathological phases, but also the disease itself, and on this point the following details throw light.

[1] Also since this paper was written four cases of aortic aneurism have passed through my hands with brief details as under :

In one, not diagnosed during life, the history was incomplete and post-mortem incomplete. In the second there was a history of syphilitic infection, rheumatism, and alcoholism. In the third (a specimen sent to the museum) the man first suffered from continued fever and bronchitis, subsequently contracted a chancre in 1871, followed by secondary syphilis, and died of aneurism in 1875. In the fourth (also sent to museum) constitutional syphilis (roseola and iritis in 1874) formed the only admissions in the " medical history sheet," the man dying suddenly in the barrack-room from rupture of the aneurism (very small, and from the sinus Valsalva) in 1875.

Throughout the pathological records I can find 117 instances of aortic deterioration, excluding those embraced under aneurism, but including both forms of the lesion already described, and the systemic conditions with which they were associated are as follows :

46·1 per cent. in undoubtedly syphilitic subjects.

6·8 „ probably syphilitic, but not beyond doubt.

21·3 „ in phthisical subjects.

14·2 „ with no record for determining the matter.

5·9 „ with heart disease.

5·7 „ with various other diseases individually small.

Here again there is a numerical preponderance with syphilitic infection ; but that which this table does not show, yet which is of immense importance in regard to aneurism, is this, that while the aortic node disease is the rule in the syphilitic diathesis, it is the exception under any other heading. For example, there are 56 cases detailed of the syphilitic virus terminating in death through special lesions, and of these 60·7 per cent. illustrate aortic nodulation and its phases, the major part of a severe type; and let this point be observed, that in about ⅓rd of the node cases (*i. e.* 18 out of the 56 cases) dilatation of the vessel, either in the form of pouching or distinct sacculated projections, had actually ensued, that is to say, were in the immature stage of aneurism, and required only further development to bring them into this classification. Adding these 18 immature aneurisms, and one subsequently mentioned as due to the acute rheumatic diathesis, to the 34 already detailed, 53 cases are at hand, and of these 66 per cent. at least occurred in subjects infected with syphilis, and with no other ascertainable systemic status. But a possible objection may be raised to these deductions in this wise ;—is not the syphilitic virus so generally dispersed in the service as to considerably weaken the inference of the connection of aneurism with it in the light of effect and cause, and rather to tend to regard these lesions as merely running side by side in the same subject ? To show how far such an

objection is valid, I have collected all the cases I can find in which the non-existence of syphilitic infection may be fairly deduced both from the previous history and post-mortem data ; these amount to 111 and give the following conclusions :

Five cases of aneurism or 4·5 per cent. ; 2 with acute rheumatic diathesis ; 2 with alcoholism ; 1 not ascertainable ; all these figure in the aneurismal list. The remaining 106 non-syphilitic subjects thus exemplify the aortic disease. In 1, or ratio of ·94 per cent. the disease was severe, and had led on to dilatation, in an acute rheumatic diathesis with alcoholism ; in 5 or 4·7 per cent. the disease had produced corrugation of the inner coat of the vessel but no dilatation—3 phthisical, 1 alcoholism, 1 aortic valve disease ; in 29 or 27·3 per cent. the disease was slight, chiefly if not wholly to be included under the second form of this paper, mere opacity of the inner wall, 15 of these instances were associated with phthisis, and the remainder with renal affections, hepatic disease, dysentery, diabetes, scrofula, lupus, cancer in nearly equal proportions.

Considering that these aortic aneurismal tumours are associated with syphilitic infection to the extent of 66 per cent., and that nodular disease of the aorta in the service is not often met with otherwise than with it, it seems an incontrovertible deduction that syphilis is a very potent cause in the production of the vascular disease and consequently also of the aneurismal tumour. The syphilitic aortic lesion in its growth, its possible stability as a fibroid node, its degeneration, its retrogression leaving a scar-like cicatrix, its impairment of normal structures in which it occurs and in its vicinity, finds its counterpart in syphilitic bone disease, cranium for example, with its surface nodes passing on to softening, cicatricial-like loss of substance, atrophy of bone elements, &c.

It must, however, be recognised that an apparently similar nodular lesion[1] of the aorta may be produced by

[1] In saying "apparently similar," I do not wish it to be inferred that I

rheumatism, alcoholism, and possibly other conditions, such as extension of disease from the aortic valves. The influence both of the rheumatic poison and alcohol on the fibrous tissues is fully acknowledged, and it can create no wonder to find them acting as irritants upon the aortic walls inducing fibroid germination terminating in fatty degeneration. That the syphilitic virus as an exciting agency of the end-arteritis is generally dispersed in the service equally as the aneurismal disease, is clear from the statistics of the secondary lesions, and I have elsewhere shown that the aortic disease is the most common sequel to severe infection in the internal structures.[1] I am also inclined to believe that although not limited to any one period of virus evolution, it is yet not uncommonly one of the earliest produced lesions, and this feature seems to explain the comparative absence of gummata in the viscera in these cases of advanced aortic disease, the aneurism killing before the so-called tertiary lesions have had time to develop themselves.

But how then does the chest constriction from accoutrements, pack, &c., stand to this theory of the connection of syphilis and aneurism ? That the aortic disease bears no relation to chest constriction and arterial obstruction, or the force of the blood expelled from an hypertrophied heart, seems to be clear from the observed post-mortem features of the disease, and the distribution of aneurism among the segments of the service. In the cases of aortic disease an enlarged heart is far from general, the disease is not at its commencement a dilatation, but on the contrary a thickening of the vessel walls,

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regard the rheumatic and alcoholic lesion as identical with the syphilitic form. That there are many points of resemblance is, to my mind, clear, though it is highly probable that attention to the subject in the future will produce features differentiating the one lesion from the other. The special character of the aortic disease due to syphilis as set forth by some writers, viz. fibroid, is a feature certainly not peculiar to it, as the lesion in undoubtedly non-syphilitic subjects is often also so in the early stage. Further evidence is required to render stable these points.

[1] Blue Book, 1870, p. 384.

and an encroachment on its area; it is true that it most frequently affects the ascending aorta, but it is far from uncommon in the transverse and abdominal portions when the ascending aorta is free; and in the few instances in which an elongation and dilatation of the ascending arch could be fairly charged to an overacting heart, no disease was present. Equally in reference to the dispersion of aneurism in the service, the statistics show it to be generally distributed irrespective of climate or occupation; the infantry man in England with his pack and full accoutrements suffers no more from the disease than does the cavalry soldier; he suffers equally as much in India and the Mediterranean with a loose special climate uniform, as in England; the cavalry man in India appears worse off than in England. But while deducing that morbid anatomy, pathology, and statistics, exclude chest constrictions as a *direct* agent, in the production of aneurism in the service, it is not intended to deny its influence *indirectly*. Given the aortic disease with impairment of the aortic walls from syphilis, rheumatism, and alcoholism as a groundwork—then the obstruction to the circulation from any chest constriction and forced exertions in full marching order must tell upon the weakened vessel and cause its dilatation when possibly no such result in the diseased vessel would ensue under ordinary conditions. The accoutrements and forced exertions of the soldier stand to aneurism in the light of fostering agencies to the germs laid by syphilis, rheumatism and alcoholism.

Equally also it may be asked how this theory stands in reference to the *excess* of aneurism in the army as compared to civil life considering that in the opinion of competent observers there is no reason to suppose that the syphilitic virus is more common to the one segment of the community than to the other? But to this it may be replied that there are no reliable data at present from which accurate deductions and comparison can be instituted. There cannot be a particle of doubt that so far as

the army is concerned, we are far from having fathomed the influence of the virus, whether in the light of a producer of disability or death, and there is nothing to guide us in civil life in gauging its true import beyond an expression of opinion. It may also be said that the practice of constantly verifying the diagnosis by post-mortem examination gives an insight into the dispersion and frequency of aneurism in the service, which the civil records do not possess, and hence comparisons cannot be fairly instituted. But granting that the amount and degree of syphilis are about equal in both communities, and consequently also an equal amount of aortic disease from it, we might infer an excess of aneurism in the army from the conditions under which the soldier is placed. Aortic disease is not necessarily followed by aneurism, but so long as the disease is present, the groundwork of the aneurism is laid, and while no dilatation need ensue under ordinary arterial pressure or such as might be present under civil exertion, yet under the forced exertions, with chest constriction, of army exercises it would be difficult to understand how such a crippled tube as a degenerate and weakened aorta could resist the extra-internal pressure. Hence the groundwork being equal, an excess of aneurism in the army might be anticipated over that in civil life, from the special conditions under which the soldier is compelled to do his duty.

We may summarise the paper as under :

(1) That in the army we have a lesion of the aortic walls characterised by the presence of a fibroid growth mainly in the internal coat, which, as a rule, ultimately disintegrates ; and that this growth is connected with syphilis in a major degree, and rheumatism and alcoholism in minor degrees, as exciting agencies.

(2) That this disease of the aortic coats may retrogress without producing any marked ulterior results upon the system at large ; but if extensive or severe, as a rule, it is followed by one of three fatal phases : formation of

aneurism, implication of aortic valve, or hypertrophy, with or without dilatation of one or more of the heart's cavities.

(3) In the army there is also a lesion of the aortic walls characterised by limited opacity or fatty change of the normal textures of the internal coat; this is common to all diseases associated with prolonged general deterioration and especially lung destruction, but it does not appear, *per se*, to lead to ulterior results.

(4) That the chest constriction and temporary forced exertions to which the soldier is liable are powerful secondary causations in the production of aneurism, acting on the portion of vessel deteriorated by syphilis, rheumatism or alcoholism.

(5) That in the adoption of preventive measures against aneurism, the attention must be primarily directed against the causes of the aortic disease, notably the suppression of syphilis, and secondarily, against the conditions of dress, &c., which assist in its development.

DESCRIPTION OF PLATE IV.

F. H. Welch on Aortic Aneurism in the Army.

Fig. 1, natural size.—A nodulated aorta slit up, cut transversely across and straightened out, to show the relative thickness of the diseased coats. (a) Internal coat extremely nodulated, encroaching on the area of the vessel; (b) middle coat normal, except opposite a, where it is somewhat thinned; (d) external coat, thickened where the middle one is thinned.

Fig. 2, × 20 diameters.—Vertical section through a node with part of the middle coat displayed. (a) Internal coat extensively thickened by laminated fibroid tissue, in which fatty or caseous degeneration has commenced in the form of granules arranged in lines; (b) middle coat normal.

Fig. 3, × 500 diameters.—A fragment of the structure of the node at an early period, the age of the particles read from left to right; from the large nucleus, through the elongated cell, to the mature fibrous tissue structure.

Fig. 4, × 20 diameters.—Vertical section through a diseased aorta, showing the condition of the internal and middle coats. (a) Internal coat thickened by laminated fibrous tissue, and still more nodulated from the presence within (b), the middle coat, of (d), a circumscribed tumour made up of delicate fibrillæ and commencing to soften and degenerate in the centre.

Fig. 5, × 20 diameters.—Vertical section through part of an ulcer of the aortic walls. (a) Internal coat thickened by fibrous laminæ; (b) middle coat with lines of fatty degeneration running through it; (d) ulcer completely eroding the internal coat, and half way through the middle one; (e) external coat which, instead of being made up of a loose meshwork, is thickened and condensed into compact fibrous tissue, evidently for the purpose of strengthening the weakened walls.

Fig. 6, × 500 diameters.—Elements forming a node in process of softening, in a state of atheroma. Granules, caseous-like particles, and cholesterine plates compose the soft mass with a few phosphatic crystals, but well-formed oil globules are decidedly absent.

Plate IV

A CASE

IN WHICH

ABDOMINAL SECTION WAS SUCCESSFULLY PERFORMED FOR INTUSSUSCEPTION IN AN INFANT SEVEN MONTHS OLD.

BY

HOWARD MARSH, F.R.C.S.,

ASSISTANT-SURGEON TO ST. BARTHOLOMEW'S HOSPITAL AND TO THE
HOSPITAL FOR SICK CHILDREN.

On the 11th of last April, while staying a night in the neighbourhood, I was asked by Dr. Miller and Dr. Barnes, of Eye, in Suffolk, to see a male infant seven months old, with intussusception. Dr. Barnes told me that on March 29th—fourteen days previously—the child had been seized with diarrhœa, sickness, and occasional griping pains in the abdomen. These symptoms, none of which were severe, and which seemed due to an attack of ordinary catarrhal enteritis, were at once relieved by small doses of castor oil emulsion, and natural feculent evacuations were passed. In two or three days, however, the griping and sickness returned, now accompanied with tenesmus; and the motions were observed to contain a considerable quantity of slimy mucus, mixed with blood. The child remained in much the same condition, and without any

marked disturbance of his general health until twelve hours before the consultation, when the whole complexion of his illness suddenly changed. His abdominal pain became very severe, sickness was frequent and tenesmus violent, and almost constant; and he grew pale and very restless.

Having been sent for, Dr. Barnes found a portion of bowel projecting two inches beyond the anus, with the ileo-cæcal valve clearly seen at the extremity of the protrusion; while, in the abdomen, a firm, cylindrical tumour could be felt, extending from the umbilicus to the left iliac fossa. A careful trial both with insufflation, and the injection of warm water into the large intestine, while the child was under chloroform, failed to reduce the invagination.

On reaching the house I found Dr. Barnes's account of the case had been so complete that there was nothing to be added to it. The intussusception was now lying just within the sphincter; but, when the child strained, it was protruded more than two inches beyond the anus. The mucous membrane was of a deep plum colour from congestion, and looked œdematous, but it was bright and glossy, and presented no abrasion of its surface. Almost at the extremity of the volvulus, but rather at the side, the ileo-cæcal opening, bordered by the two nearly horizontal cusps of the valve, was distinctly seen. The child's napkin was stained with serum and blood, which oozed in considerable quantity from the mucous membrane of the invaginated bowel. In the abdomen a firm, sausage-shaped tumour could be very readily felt; indeed it could be plainly seen when the child, in straining, tightened his muscles. Its upper end, abrupt and well-defined, was placed just to the left of the umbilicus; and from this point its length could be traced in the course of the descending colon and rectum down into the lower part of the pelvis. The child lay in his nurse's arms retching every two or three minutes; with a pale face, sunken half-closed eyes, a small and very rapid pulse, and shallow,

hurried respiration. The parents begged that whatever gave a reasonable hope of saving his life might be done.

When he was under chloroform the abdomen. was opened by an incision about two inches long, beginning in the middle line, immediately below the umbilicus, and running downwards towards the pubes. It was found that when the recti had been pulled aside the subjacent tissues, including the peritoneum, were so thin that much care was needed to avoid injuring the intestine, which bulged forward into the wound. Two fingers were now passed down to the upper end of the intussusception, the position of which was already precisely known, and an effort was made to draw out the intestine. This quite failed, and the impression left on my mind at the time was that such an attempt would be very little likely to succeed, except, perhaps, in the case of a very short intussusception; for with only two fingers it was impossible to fix the tumour, and, at the same time, to pull out the volvulus. I therefore brought the upper end out through the wound, and gently dragged at the entering portion of the gut. For a moment it would not come, but, using a slightly increased force, I felt that its distal end was started, and then it glided smoothly up the colon, the canal of which seemed large enough to let it pass along with the utmost facility. At last the cæcum and its appendix emerged, their serous surface being clearly presented to view. What length of intestine had been invaginated cannot be accurately stated, but obviously this included at least half the colon and an equal portion of the small intestine. When the intussusception had been reduced, a considerable proportion of the intestines, with a part of the great omentum, lay on the surface of the abdomen; and the return of these coils through an opening even two inches long proved to be by far the most difficult part of the operation ; but with Dr. Barnes' help, and while Dr. Miller kept the child still under chloroform, this was accomplished without much delay. The wound was then closed with two harelip pins and

some superficial sutures, and supported with long pieces of strapping and a bandage.   There was no bleeding of any importance during the operation.   The case was subsequently entirely under the care of Dr. Miller and Dr. Barnes.   Small doses of laudanum were given three or four times in the course of the next twenty-four hours ; and the child drank freely of milk and water, to which, when there seemed occasion, a few drops of brandy were added.   The sickness ceased immediately after the operation, and did not return ; flatus and a small quantity of blood-stained mucus were passed from the bowel twice or three times in the first twelve hours; the child had no pain, and the abdomen remained soft and free from tenderness.

On the 13th Dr. Barnes found the child bright and playing ; without pain or fever, and with a pulse of 95.

On the 14th he had slept almost all night.   A feculent motion was passed early in the day.

On the 15th the pins were removed, eighty-four hours after the operation ; the wound was found nearly healed, and its edges showed no tendency to separate.   From this time there was nothing to report except that, in a few days, the child had perfectly recovered.   He is still in good health.

Remarks.—The chief features of interest in this case will be placed in the strongest light if, instead of discussing them alone, they are examined in their bearing on Mr. Hutchinson's paper on intussusception, published in the 57th volume of the Society's ' Transactions.'   Before that communication was read it was the very general opinion that abdominal section ought not to be performed in cases of intussusception, because, among other reasons, the intestine became so damaged by inflammation and strangulation, and so fixed by the mutual adhesion of its different layers, that it could not be safely drawn out. But Mr. Hutchinson directed particular attention to the fact—which, though it had been often noticed before at post-mortem examinations, had never been credited with

much clinical importance—that, in many cases, the condition of an intussusception is not that of inflammation, strangulation, or adhesion of its layers, but of simple invagination ; the volvulus lying loose in its sheath, in a state comparable with that of a portion of intestine prolapsed into a hernial sac ; but through which the circulation is still free, and surrounding which there are no adhesions. The case just related bears out this observation in a striking manner. The child's illness was divided into two distinct periods ; the first—extending to the early part of the fourteenth day—in which all the symptoms were so chronic that they raised no suspicion as to the real nature of the affection ; the second—occupying the twelve hours immediately preceding the operation —in which all the symptoms were very acute. Doubtless the onset of these acute symptoms marked the time at which mere invagination was succeeded by inflammation and strangulation. And what was found at the operation corresponded with this view. No firm adhesions were discovered ; but in drawing out the intestine just so much resistance was at first encountered as might be due to oedema, and commencing adhesions, resulting from twelve hours of acute strangulation. Difficult or even impossible as it may be in, perhaps, the majority of cases, to feel any certainty as to the condition of the intestine, it seemed in this instance safe to conclude that it had not been materially damaged.

Mr. Hutchinson refers to the effect which the age of the patient is likely to have on the result of the operation ; and he remarks that " in the absence of any data as to the manner in which operations of this kind are borne by very young children, we shall probably be right in believing that they are far less hopeful than in those that are somewhat older." The evidence derived from this case, however, points the other way, for though the patient was only seven months old, though he was in a condition of collapse at the time of the operation, though the intussusception was very extensive, and though a con-

siderable length of the intestine lay at one time on the surface of the abdomen and was not returned without free handling, yet recovery took place without a bad symptom, and with no more trouble or suffering than usually attends an operation for harelip; and the patient, Dr. Miller and Dr. Barnes informed me, was convalescent on the fourth day.

In recording this case I must beg permission of the Society to add that I am far from advocating that abdominal section should be performed indiscriminately in cases of intussusception. Such a course would inevitably tend to bring surgery into disrepute. But that there are instances in which the operation should be done is proved by Mr. Hutchinson's case and by that related above.

If the diagnosis is certain, and if other means, carefully tried, have failed, my conviction is that the operation ought at once to be performed; *first*, in cases in which strangulation is acute and quite recent; that is, of not more than twelve or, at the most, eighteeen hours' duration. *Secondly*, in cases which are chronic, and in which there have been no symptoms of inflammation or strangulation. The responsibility of operating must always be great and failures must be expected; but I believe the responsibility of withholding the operation in all cases will be greater still.

A CASE

OF

INTUSSUSCEPTION IN AN ADULT,

WITHOUT SYMPTOMS OF STRANGULATION,

TREATED SUCCESSFULLY BY ABDOMINAL SECTION.

BY

C. HILTON FAGGE, M.D.,

ASSISTANT PHYSICIAN TO, AND LECTURER ON PATHOLOGY AT,
GUY'S HOSPITAL,

AND

HENRY G. HOWSE, M.S.,

SURGEON TO GUY'S HOSPITAL AND TO THE EVELINA HOSPITAL FOR SICK
CHILDREN.

THE following case is of interest, as showing the value of abdominal section, or laparotomy, as some recent writers prefer to call it, in those cases of intussusception which inflation has failed to cure.

. Elizabeth M—, æt. 33, residing at 16, New Street, Bermondsey, applied to Dr. Adcock, of Abbey Street, on June 13th, 1874, on account of a spasmodic pain in the abdomen, just above the umbilicus. She was ordered a mixture containing morphia and aqua anethi. This, however, afforded her no permanent relief, and she continued to attend at the surgery until the 20th. On that day her symptoms were sufficiently urgent to lead Dr. Adcock to advise her to go home and to go to bed, and to take one grain of opium every four hours.

On the 21st Dr. Adcock visited her at her home, and for the first time examined her abdomen. In the right iliac fossa he found a swelling of about the size of a hen's egg, but not very clearly defined. The bowels were open,

but the pain was not much relieved. The opium was now discontinued. She remained in bed until the 23rd, when, feeling better, she got up ; but on the following day she was obliged to go to bed again on account of violent pains about the umbilicus. From this time her condition varied ; she was sometimes better, sometimes worse. The tumour gradually moved over into the left iliac fossa.

On the 28th Dr. Fagge was asked to see her in consultation with Dr. Adcock. She was then lying in bed. She complained of pain, which came on at intervals, and which she referred chiefly to the umbilicus. She had had some sickness; the vomited matters were not stercoraceous, and, indeed, presented no peculiarities worthy of note.

On examining the abdomen Dr. Fagge was at once struck with the resemblance between the tumour, which could be felt in the left iliac fossa, and that which is observed in cases of intussusception. It formed an ill-defined mass, elongated in the direction of the descending colon. No peristaltic movements could, however, be felt in it : nor did it at that time harden under manipulation. Still, the fact that it had been originally felt in the opposite iliac fossa supported so strongly the view that it really was an intussusception that this view was laid before the patient, and a proposal was made to inflate the bowel. She readily consented, and the nozzle of a pair of bellows was without delay introduced into the anus and air was pumped into the gut. There was no apparent result from this procedure.

On the following day (the 29th) Dr. Fagge saw her again; and at his request Mr. Howse was asked to meet him and Dr. Adcock. Mr. Howse, after examining the patient, agreed in the diagnosis; and inflation was performed for the second time, but with more perfect instruments than at first, so that the air could be pumped in with greater force. It gave her very great pain, and therefore they thought it prudent to desist, before the full power at command had been exerted; the tumour became exceedingly hard at this time. Afterwards it

appeared to be situated at a higher level than before, and to be somewhat softer. In the evening, however, it was found to have disappeared entirely from the left iliac fossa, which could be explored thoroughly and was perfectly empty. The tumour now lay behind the umbilicus, passing across to the right side.

On the 30th inflation was practised for the third time, but with no result. The patient said she felt much better. Her temperature was 99°, her pulse 95. The tongue was clean, as it had been all along. She had had no sickness since the second inflation. Her bowels had not been opened.

On July 1st her symptoms remained almost unaltered. She had had a slight return of sickness in the morning and a little pain. Her pulse was 94; her temperature normal; her tongue clean. The bowels had not been opened.

The hard mass could still be distinctly felt lying behind the umbilicus.

We both, as well as Dr. Adcock, felt perfectly confident about the diagnosis; and the question was now very anxiously discussed whether inflation of the bowel should be repeated, or whether an exploratory operation should be performed. And as inflation had been attended with only partial success, and had given rise to intense pain, fears were entertained lest the repetition of this procedure should lead to rupture of the bowel. The matter was thoroughly explained to the patient. She was told that an operation would afford her a good prospect of recovery; but that, if it were not done soon, fresh symptoms might be expected quickly to develope themselves, which would greatly diminish the probability of success, if the operation should afterwards be undertaken. Both she and her husband readily gave their consent.

Chloroform having been administered, the patient was accordingly placed on the table. All the established precautions of the antiseptic system being in force, an incision about three inches long was made opposite the

umbilicus, and the various layers down to the peritoneum were incised just as in ovariotomy. A small opening was then made into the peritoneum, and a finger immediately inserted so as to plug the aperture. The remainder of the peritoneum was incised upon this finger, and as the aperture was enlarged the other fingers of the same hand were inserted in order to keep the aperture closed. In this way the whole hand was introduced into the peritoneal cavity. But before this could be done it was found necessary to extend the incision to four inches in length. The precaution of plugging the wound was taken to prevent undue quantities of carbolic lotion from flowing into the peritoneal cavity. The hand was then passed into the right iliac fossa, where the intussuscepted mass was readily made out. It was hoped that it would have been possible to reduce the intussusception *in situ*, but all attempts to effect this failed. The intestine was accordingly drawn out from the abdominal cavity and placed on the surface of the abdomen. Even then some difficulty was experienced in reducing it. Pulling at the ends was quite ineffectual, though as much force as was considered justifiable was exerted in this way. Under a kind of kneading movement, however, combined with circular pressure upon the farthest intussuscepted part of the gut, it began to yield, and when once started the process went on readily, until the last part of the intestine was reached. Here a slight check took place, but this again was speedily overcome. More than eighteen inches were in this way drawn out. The bowel was then quickly restored to the abdominal cavity. No lymph was found on the serous surface of the gut, which was quite smooth and shiny. As far as could be seen (for the whole proceeding only occupied a few minutes), the intussusception was of the usual kind, consisting of an involution of the lowest end of the ileum and the beginning of the large intestine into the succeeding part of the colon. During the time that the intestine was outside the abdominal cavity, care was taken to keep it covered with a large

double fold of lint and flannel, soaked in warm carbolic lotion, of a strength of 1–40, so that only just the portion of the intussusception under immediate manipulation should be exposed at one time. The wound was brought together with interrupted sutures in the way practised in ovariotomy, serous surface being applied to serous surface. The deep sutures, viz. those passing through the peritoneal surfaces, were of silk, soaked in carbolic lotion and oil. The superficial sutures, for bringing the edges of the skin together, were of antiseptic gut. The wound was then dressed with two layers of antiseptic gauze, each of eight thicknesses, the first layer soaked in carbolic oil (1–20), the second dry. The carbolic spray (with lotion of 1–40) was kept playing until the dressing was complete. A suppository of morphia was administered immediately after the operation.

On July 2nd the patient was very comfortable, save a certain amount of chloroform-sickness. She was cheerful and almost free from pain. Pulse 98; temperature 98·3°; tongue clean. The bowels had acted slightly, and urine had been passed freely.

On July 3rd the pulse was 112, but the temperature was normal. Tongue clean. She was still very sick, and she was accordingly ordered a mixture containing morphia, bismuth, and prussic acid, to be taken every four hours.

The next day the sickness had abated. She was, however, still kept on milk, ice, and soda water. From this date she rapidly improved. Some of the superficial sutures were removed on the fourth day, and two of the deep ones on the sixth day, but all the sutures were not finally withdrawn until the tenth day. Complete primary union had then taken place along the whole course of the wound, but the dressings, strapping, and bandage were kept on for another week, so as to support the line of incision and allow complete consolidation to take place. After this period a bandage was the only support which the patient wore. The temperature, throughout, never rose above normal, and the bowels acted regularly after

the fourth day. We saw her for the last time at home rather less than three weeks after the operation, but she has once or twice since been up to the hospital either to show herself or to be treated for different ailments. At the present time (November 29th) she is quite well and awaiting her confinement.

In a paper on Intestinal Obstruction, written by Dr. Fagge for the 'Guy's Hospital Reports,'[1] it was remarked that the progress of an intussusception may in many cases be divided into two stages. One of them was defined as being characterised by the presence of a tumour, and by attacks of severe pain, recurring at intervals, between which the patient might appear quite well. The other stage was regarded as coincident with the occurrence of strangulation in, and with the commencement of the changes which lead to the death of, the intussuscepted part of the bowel; the indications of its supervention were said to be that the pain became more or less constant, and that anxiety and constitutional disturbance were present, as well as hæmorrhage from the bowels and tenesmus.

Mr. Jonathan Hutchinson, in his paper read before this Society, in 1873, on the Treatment of Intussusception by Abdominal Section, insisted on the importance of the distinction between those cases of intussusception in which the included part of the bowel is strangulated, and those in which it is not. But he regarded them as belonging to two different forms of the disease, not merely to successive stages of it; and he did not place hæmorrhage from the bowels, nor tenesmus, among the symptoms indicative of strangulation.

Now, it appears to us that neither Mr. Hutchinson's views, nor those which were laid down in the paper in the 'Guy's Hospital Reports,' correspond altogether with the facts.

On the one hand, it is certain that in many cases,

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[1] Vol. xiv, 1869, p. 272.

even when the disease runs a chronic course, hæmorrhage is present from the first. Thus in Mr. Hutchinson's case, in which he successfully performed abdominal section, the first attack of pain in the abdomen was quickly followed by a motion containing blood; and the child continued to pass blood-stained mucus for a month. Yet at the operation the intussuscepted mass was pulled out without difficulty, and the opposed serous surfaces· did not present a single flake of lymph, and were even congested only in very moderate degree. It is therefore clear that the occurrence of hæmorrhage is not an indication of the commencement of the process of sloughing, and casting off of the invaginated part of the bowel.

But on the other hand, there are cases of intussusception which run on for a great length of time without any blood being passed from the bowel; and in which, when hæmorrhage once occurs, it is accompanied by symptoms of severe constitutional disturbance, which rapidly destroy the patient's life.

A striking instance of this, which came under the observation of Dr. Fagge some years ago, is recorded in the paper to which we have already referred. A child, æt. 5, was admitted on account of an intussusception which had already existed four months; the symptoms were paroxysmal attacks of pain, and the presence of a sausage-like tumour above the umbilicus. The nature of the disease was diagnosed three weeks after admission; but no active interference whether by inflation or operation was attempted, it being feared that adhesions had formed which it would be dangerous to disturb. A week later the child began to pass blood and mucus; and in three days it died. The post-mortem examination showed that there were indeed shreds of lymph between the entering and returning layers, but that there was no ulceration nor gangrene, although the entering layer was much swollen and thickened.

In such a case as this it is natural to speak of the invaginated ·mass as "strangulated," and to regard the

hæmorrhage as a symptom of the occurrence of "strangulation." But we must consider what exactly is meant by such a use of the term. Now, bleeding from the intestinal mucous surface does not commonly take place in strangulated hernia, nor when a loop of intestine is constricted by a band, although in either case the affected portion of the bowel is very apt to become ulcerated, and even gangrenous. The reason doubtless is that in those forms of disease the neck of the strangulated gut is so forcibly compressed that all flow of blood into it through its arteries is quickly arrested after the strangulation has once commenced. But in an intussusception it may well be that the return of venous blood from the invaginated mass is obstructed for a long time before the pressure is sufficient to cut off its arterial supply. And this appears to us to be the real explanation both of the occurrence of hæmorrhage in such cases, and of the great swelling which is well known to occur in the included part of the bowel.

It seems, therefore, that instead of hæmorrhage being proof of sloughing in a case of intussusception, it actually indicates that the affected part of the bowel is in a different state.

And, in looking through the list of twenty cases of intussusception followed by the shedding of part of the bowel, which Dr. Peacock has collected in the 15th volume of the 'Pathological Transactions,' we find that there is only one out of the whole number in which it is stated that blood was contained in the evacuations; even in that one instance hæmorrhage occurred only at the commencement of the disease, and not for ten or twelve days before the date at which the cast-off portion of intestine came away. It is true that blood is sometimes passed with the sloughing bowel; but this seems to be less frequent than is generally supposed, and it is doubtless the result of ulceration at the neck of the intussusception.

It is important to note that the supervention of severe constitutional disturbance is not, any more than hæmor-

rhage itself, an indication of the occurrence of gangrene. In the case of the boy, æt. 5, already mentioned, death followed within three days from the time when blood was first present in the evacuations. The symptoms during this period were constant pain, vomiting, restlessness, fever, a quick and very feeble pulse, and rapidly increasing distension of the abdomen. But, as we have already stated, the invaginated mass was not sloughing, nor even ulcerated. Indeed it seems to us, after having carefully looked through the notes of the symptoms which were present in the cases collected by Dr. Peacock, that there are not any symptoms which really do indicate the occurrence of gangrene in the included bowel. Cases in which this takes place seem generally to be characterised *rather by the absence of those symptoms which are peculiar to intussusception as compared with other severe intestinal lesions.* There is, indeed, one point which, as Mr. Hutchinson has remarked, positively indicates that sloughing has *not* commenced ;—viz. the fact that the tumour continues to advance further and further in the direction of the large intestine. For such an increase in size is well known to involve a gliding movement in the parts which form the neck ; more and more of the receiving part of the bowel becoming included within the invaginated mass ; and such movement is utterly incompatible with the processes which accompany gangrene.

We have already observed that in many cases hæmorrhage from the bowels is attended with severe constitutional disturbance, and leads rapidly to a fatal issue. These symptoms appear to be the effect of the extreme congestion and swelling of the intussuscepted mass, which take place under such circumstances, and which, as is well known, reach their maximum at its *lower extremity,* or the part which is farthest from its neck.[1]

One consequence of this change in the included part of

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[1] Dr. Moxon mentions in his work on Pathology that he measured the coats of the bowel in one case at this part, and found them ¼ of an inch thick.

the bowel is that there is sometimes great difficulty in effecting complete reduction of the intussusception, even at a post-mortem examination. In a case in which our colleague, Dr. Goodhart, made an autopsy in 1873, and in which death had occurred within two days from the commencement of the disease, he found it impossible to replace the whole of the bowel. Attempting to draw out the invagination led to no result, except that the peritoneum became torn. Squeezing the mass was then tried, and this had the effect of forcing out all but the pouch of the cæcum, which still remained inverted in spite of all the force which could be used, forming a cup-shaped depression on the serous, and a small rounded eminence on the mucous surface of the bowel.[1]

It is therefore evident that the occurrence of hæmorrhage from the bowel may be accompanied by, and be indicative of, the supervention of changes in the intussuscepted part, which would interfere with the success of an operation for the cure of the disease, or even of inflation of the bowel with air. And if the disease can be dia-

[1] September, 1876.—Since this communication was read before the Society another case has come under our observation, which shows that the changes in the lower end of an intussuscepted portion of intestine may be still more considerable. A child, æt. 5 months, was brought to the hospital, who had for a month been suffering from abdominal symptoms, and in whom the ileo-cæcal valve had been protruding from the anus for some days. Mucus and blood had been discharged in considerable quantity. The medical man who had been in attendance had mistaken the case for one of simple prolapse of the rectum, and had contented himself with returning into the bowel the extruded mass. The child came under the care of Mr. Howse, who asked Dr. Fagge to see the case with him. Inflation being out of the question, abdominal section was at once performed. A large part of the intussusception was reduced without difficulty; but the last four inches, close to the ileo-cæcal valve, were firmly adherent and so softened by inflammatory action that traction, although most gently performed, caused two considerable rents, from which fæces escaped. As it was clearly hopeless to return the bowel in that condition into the abdominal cavity, the mass which remained intussuscepted was cut away altogether, and the portions of healthy intestine above and below were sewn together. The abdominal cavity was then closed, but the child survived only a few hours. A fatal termination was, in fact, altogether inevitable in this case, whether left alone or treated in whatever way.

gnosed with certainty before this symptom shows itself, it is clearly important that this should be done. From this point of view we think that our case possesses considerable interest.

But perhaps the question may be raised whether the symptoms that were present in that case—paroxysmal pain and tumour—were really sufficient to justify the diagnosis that the disease was intussusception. And hesitation in regard to this point would be the more natural, because such disease is of rare occurrence in grown-up persons. After an experience of many years Dr. Wilks stated that he had seen only one instance of intussusception in an adult. It seems to us, however, that the characters and position of the tumour were of themselves conclusive as to the nature of the case. We were informed that the swelling, when first noticed, had been situated in the right iliac fossa, and that it had moved over to the left, and after inflation we observed that it was absent from the position that it had before occupied, and had passed back towards its original seat. This was proof that the tumour was in the intestine. The only doubt that could remain was whether it could possibly be an accumulation of fæcal matter or of undigested food. There is a case of Dr. Brinton's on record in which a mass of half-chewed filberts formed a swelling of the size of a pullet's egg, situated at first in the right hypochondrium, but which afterwards passed down into the right iliac fossa, and finally disappeared. And in 1870 Dr. Fagge met with a case in which intussusception was suspected, but which proved to be simply one of impacted fæces. A woman, æt. 22, was admitted into the Clinical Ward. She had been seized twelve days before with pain in the limbs and constipation. She had taken an aperient and the bowels had acted twice. After this she had had no motion up to the time of her admission. The abdomen was then flaccid and free from distension; there was no tenderness. The rectum was empty. She had vomiting after food. Subsequently a

tumour was felt to the right of the umbilicus, varying in position and in degree of hardness. Two or three days later a hard cord was discovered in the position of the descending colon and sigmoid flexure. The rectum now contained fæces. An injection was therefore administered, and it brought away some scybalous masses. Eleven days after her admission another large injection was prescribed. The clinical clerk threw up seven pints, the result being that a large quantity of hard fæcal matter came away. The patient, however, became severely collapsed, breathing rapidly and with distress. It appeared that due care had not been taken in the administration of the enema, and that the fluid had been injected more rapidly than was right. She was now ordered six ounces of brandy, and she rallied somewhat. But the bowels continued to act; and she became convulsed and died about six a.m. on the following day. At the autopsy the rectum, as far as the anus, was found to be loaded with round fæcal masses. There was no fluid feculent matter in any part of the intestine. There was no intussusception nor any cause of obstruction except the accumulation of fæces. This had, no doubt, been diminished by the injection, the incautious administration of which appeared to have led to the patient's death.

In that instance, as we have already remarked, it was thought possible before death that an intussusception existed. But the tumour was very far from presenting the definite characters that existed in the case that is the subject of the present communication. In the latter not only did the swelling move from the right to the left iliac fossa and afterwards recede, but its size and hardness underwent variations under manipulation; when a paroxysm of pain occurred it became very much more resistant than before. This peculiarity of the tumour formed by an intussusception was particularly noticed by Dr. Fagge in another case, to which we have already[1] made

some reference, and in which the patient was a boy, five years of age. In that instance the swelling sometimes could not be discovered when the hand was first placed upon the abdomen, but in the course of the examination it would become hard and prominent. More than one writer has described the same thing. In his well-known paper in the 'Mémoires de l'Académie de Médecine' for 1860, Duchaussoy quotes from the 'Archives Générales' (1836, tome xii, p. 240), the account of a case by Dr. John Wood, in which, "when the abdomen was examined, one could at first discover only a fulness and an abnormal resistance in the right iliac region; but if the hand was retained in this position while an attack of pain occurred, an oblong tumour could be felt, which swelled up as if by a *process of erection*. At the same time a gurgling sound was heard. . . . . ." Gasté again (according to Duchaussoy) saw the tumour in his patient increase in size with each new spasm; Nissen felt such a tumour descend; and Phelan observed one which had a vermicular movement that was readily to be recognised. Dr. Brinton met with a case in which there was a long cylindrical tumour, bent into a curve with its concavity upwards, and which was the seat of an active writhing peristalsis.

The only other point that appears to require comment is our having so quickly given up the attempt to reduce the invagination by inflation, and at once submitted the patient to the operation of abdominal section. With regard to this we would observe that the injection of air into the intestine is itself not unattended with danger. In November, 1873, Mr. Howse was asked to see a patient of Dr. Wilks', a child æt. 6 months, in whom the intestine had been ruptured and the peritoneal cavity distended with air, as a result of attempted inflation. It had been suffering for less than twenty-four hours under the symptoms of intussusception, including hæmorrhage from the bowels. Dr. Wilks had directed the performance of inflation, and this had been done shortly after the

termination of his visit by the house-physician and the clinical clerk. The abdomen had at first become irregularly distended, but afterwards it suddenly became uniformly full all over. Air then no longer escaped from the rectum, and the child was almost pulseless. Mr. Howse was then sent for with a view to the performance of abdominal section, but the case was evidently unsuitable for the operation. To relieve the distension the peritoneal cavity was punctured with a fine trocar and canula, and a large quantity of air was allowed to escape, but the case soon terminated fatally. At the autopsy it was found that there was much fæcal matter in the abdomen. The peritoneal coat of the bowel had given way at one spot over an area of the size of a split pea; and in the centre of this there was a minute opening in the other coats, through which the contents of the bowel were escaping. In two other places the serous covering of the intestine was alone torn through. And in the case of another child, æt. 7 months, in which inflation had first been used with partial success, and afterwards injection of water, Dr. Moxon found that the colon had partially given way in several places. The peritoneum was cracked through, and the edges of it were folded back and fixed by agglutination in their new positions. One part of the bowel of some length presented a row of transverse fissures corresponding with the sacculi. These extended through the longitudinal muscular fibres, whereas they formed gaps between the transverse fibres.

In the case which is the subject of the present paper the patient complained of great pain while we were working the bellows, especially on the second occasion; and we came to the conclusion that no further good would result from that procedure. Indeed Mr. Hutchinson has summed up both his own opinion and that of the best authorities in the statement that all attempts at replacement of the bowel by injections of fluid or air ought to be abandoned if they do not succeed within a short period.

NOTES OF A SECOND CASE OF

ABDOMINAL SECTION

FOR

INTUSSUSCEPTION INTO THE COLON,

WITH

REMARKS ON THE DETAILS OF THE OPERATION.

BY

JONATHAN HUTCHINSON, F.R.C.S.,

SENIOR SURGEON TO THE LONDON HOSPITAL; SURGEON TO THE ROYAL
LONDON OPHTHALMIC HOSPITAL, MOORFIELDS; AND TO THE
HOSPITAL FOR DISEASES OF THE SKIN.

A SECOND case of operation for intussusception, which occurred to me a few weeks ago, gave me the opportunity of acquiring additional experience as regards some points of detail which it is, I think, of importance to bring before the profession.

The patient was an infant, aged 6 months, under the care of Dr. Madge, by whom I was asked to see the case on the fourth day of the illness. The symptoms had from the first been well-marked, the child having experienced frequent attacks of painful straining with the passage of bloody mucus. The invaginated part could be easily felt in the left flank by manipulation through the

abdominal wall, and also by the finger introduced into the rectum. It had not been actually extruded, but its lower end was often within an inch of the anus. Dr. Madge had fully diagnosed the case before I saw it, and the usual measures of treatment by injection, &c., had been carefully employed. An infant sister of the child had died of the same lesion about a year previously. When I saw the infant it was evident that there was no time to be lost, for it had rejected all food for three days. It was very weak and did not seem likely to live many hours ; we therefore persuaded the parents to allow us to perform the operation at once. I found the operation, as perhaps might be expected at so early an age, much more difficult of accomplishment than in my former case, and it is chiefly to these difficulties that I wish to ask the attention of the Society. The abdominal wall was thick, being loaded with fat, and the space between the umbilicus and pubes was very short. Having made an incision in the *linea alba,* just sufficient to admit two fingers, I was able easily to reach the neck of the intussusception, but I found it so slightly moveable that I could not by any means hook it into the wound. I was very anxious not to allow the intestines to escape through the wound, and finding that I could draw the bowel out to a certain extent between the tips of my two fingers, I spent some time in trying to accomplish reduction in this manner, hoping to complete it without exposing the bowel to view. At length, however, I became convinced that this was impracticable ; only a certain length could be drawn out and then it became firmly fixed. I was obliged, therefore, to enlarge the wound freely above the umbilicus, and to allow the intestines, much distended with gas, to escape. It was only when the abdomen was almost empty that I could bring the neck of the sac into view in the wound, and I then made repeated attempts to draw the bowel out, but without success. That there were no adhesions was proved by the fact that an inch or two could be easily drawn out ; the impediment was clearly due to the

ensheathing bowel being thrown into folds by traction, and thus constituting a series of strictures which gripped its contents. In this dilemma, and when almost in despair as to whether I should accomplish the reduction, it occurred to me to seek the lower end of the invaginated part, and try to hold the ensheathing layer so as to prevent its being drawn into folds. This led to the discovery that it was only the neck of the invaginated tract that was in any degree fixed (by its mesentery); the lower part, consisting of the sigmoid flexure of the colon much elongated, lay in loose folds on the rim of the pelvis, and was hooked out of the wound with the greatest ease. The attempt to hold the ensheathing layer straight at once revealed the true method of reduction, for by pulling this downwards, instead of trying to pull the involved part upwards, I accomplished the replacement with the greatest ease. The *appendix vermiformis* came out last, just as the reduction was completed, proving that the intussusception had begun at the cæcum. The operation had been protracted and the infant was almost pulseless, but there still remained the task of replacing the intestines within the abdominal cavity. I had some difficulty in accomplishing this, and several times after their return, portions escaped again before I could close the abdominal wound. Under these circumstances I was induced to prick the distended small intestine at two or three places with a harelip-pin to allow the escape of flatus. To my chagrin, however, just as I was closing the abdominal wound I observed that one of the punctures in the bowel was bubbling, and that there was a stain of feculent matter on the peritoneal surface; this was carefully wiped away and the wound closed. The child, although in extreme collapse when the operation concluded, rallied subsequently, became warm, took the breast, and passed a feculent motion. Death, however, took place in the night, about eight hours after the operation.

At the *post-mortem* next day we found no trace of any

further escape of fæces, but there was almost universal peritonitis, the coils of intestine being glued together by lymph. It became of interest, as one of the child's sisters had died with precisely the same condition, to ascertain whether there was any congenital peculiarity which might account for its occurrence. We found the parietal peritoneum in the right flank quite smooth and entire, whilst the cæcum hung very loosely attached by a long mesentery. This had probably been much stretched in the process of invagination, but there could I think be no doubt that the cæcum had been loose congenitally. No reproduction of the invagination had occurred.

It is my impression, judging from my experience of this case, that in future operations the lower end of the invaginated tract ought always to be first sought, and that reduction ought to be accomplished by squeezing it or pulling the sheath downwards rather than by attempting to pull the contained tube out. I am not at all sure that in some cases this might not be accomplished without bringing the parts into view; should this not be found practicable, however, it is probable that the operator will find it much more easy, in cases of intussusception into the descending colon, to bring the lower part into the wound than the upper one. It is the more necessary to draw attention to these points because they are both probably unlikely to occur of themselves to the operator's mind.

NOTE

ON

PATHOLOGICAL ABSORPTION SPECTRA.

BY

EDWARD LAWTON MOSS, M.D., F.R.C.S.I.,

SURGEON TO H.M.S. ALERT.

(COMMUNICATED FROM THE DIRECTOR-GENERAL OF THE MEDICAL DEPART-
MENT OF THE NAVY BY

JOHN COOPER FORSTER, F.R.C.S., HON. SEC.)

LIKE many other animal fluids, urine absorbs the
shorter light waves indiscriminately. The absorption is
of course in proportion to the thickness of the stratum of
fluid examined. If the stratum is too thick, or the tint
too deep, any delicate characteristics which may exist are
masked. An ordinary test tube about ¾ inch bore holds
a convenient thickness of urine for absorption observa-
tion. The intensity of the light, and the dispersive
power of the spectroscope, are also important preliminary
considerations. An instrument with a single water prism
or one of Browning's convenient little direct-vision spec-
troscopes will give better results than a more powerful
apparatus, for a faint band of absorption is not easily seen

when it is dispersed over a large part of the field of sight. The great range of colour possessed by various urines does not display an equally conspicuous diversity of absorption spectra, and the complicated chemical colouring matters, such as uropittine, paramelanine, or omicholine, fortunately bring none of their confusion into spectroscopic operations of fresh urine.

Other fluids mixed with urine give of course the absorption characteristics of their colouring matters, and thus the spectroscope yields the earliest evidence of the hæmaturia of Bright's disease. In a case of that affection recently under my observation, the Sorby blood bands in the urine preceded any definite identification of albuminuria by the ordinary tests, though the daily quantity of urine was already increased. In this case one retina only presented the white extension from the disc, and it was so slight as to require careful focussing to display it.

The existence of blood bands in the absorption spectrum of urine gives far more definite evidence than mere smokiness, albuminuria, or even the subsidence of shrivelled and altered corpuscles, and their presence or absence in albuminous urine, may throw some light upon the capricious way in which the proteine compounds act when subjected to the so-called albumen tests.

From time to time during a series of observations now unavoidably interrupted I came across absorption bands in various parts of the spectrum—generally in the yellow and green—which were of temporary occurrence, and which I failed to connect with any other phenomenon. A case of cirrhosis of the liver, however, supplied a urine which, for the three weeks I had the opportunity of watching it, gave a permanent absorption band of an interesting character. This band is not mentioned in Dr. Thudichum's exhaustive paper in the Tenth Public Health Report; I therefore suppose it to be novel, though I have not been able to obtain the results of Hoppe-Seyler or Valentin's inquiries. This case in addition to

the ordinary urinary absorption, diffused more or less equally over the whole blue end of the spectrum, exhibited a well-defined and intense band (see Plate V) lying between 1700 and 2100 of Kirchoff's scale, and thus just including the F solar line in its right edge.

The band became more intense as the urine became staler and more acid. Boiling also intensified it, irrespective of any condensation of tint from evaporation. It was also darkened by the addition of acid. Nitric acid upon some occasions brought out a faint additional band, lying its own width to the right of the sodium line in the position of a deoxidized blood band. Permanganate of potash, added in quantity less than sufficient to produce its own complicated bands[1] permanently, also increased the absorption in this spot. It naturally suggested itself that this band might have something to do with the existence in the urine of biliary products. Pettenkofer's test did not yield the cholic-acid play of colours, though a change from orange into purplish magenta, caused probably by the action of the acid used on biliary colouring matter, might have been mistaken for it (the acid was acetic, used on Lehmann's authority). It is remarkable that the band in question was very conspicuous in this magenta solution and remained so for more than a week; its right edge, however, was more distinct than in the original urine. Various experiments on the bile of such animals as I could procure, including ox, sheep, deer, racoon, &c., failed to produce any similar absorption band. Meconium, which, according to Simon and Frerich, is almost unchanged bile—was the nearest approximation to human bile available; it also failed to yield it, however manipulated. I at length found it in normal acid fæces; the absorption of a fæculent solution occupied precisely the same position and behaved in every respect like the

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[1] The conspicuous way in which the spectroscope told the exact point of saturation of the urine with potash permanganate suggested its usefulness in many instances of volumetric analyses in which the saturation tint is not sharply defined.

abnormal urine band. It was altogether absent in healthy alkaline fæces, but appeared almost invariably on keeping on acidulation, or on the addition of potash permanganate. This band in the spectrum of urine and fæces disappeared on neutralisation with ammonia, and reappeared on reacidulation. The substance yielding it seems to be some free acid. It is soluble in alcohol but not in ether.

The foregoing notes are altogether fragmentary, and have been put together in unavoidable haste, but they may in some humble measure tend towards the eventual application of the spectroscope to practical medicine—an application which appears to me capable of deciding, for example, whether the high-coloured urine of a tropical pyrexia owes its tint to the altered bile of bilious, or the disorganised blood of yellow fever.

DESCRIPTION OF PLATE V.

Morbid urinary absorption spectrum occurring in a case of cirrhosis of liver. In addition to the obscuration of the blue, indigo, and violet which urine generally produces, there is a band of intense absorption occupying the space between 1700 and 2100 of Kirchoff's scale, and covering the F solar line in its right edge.

A similar absorption band is frequently yielded by normal acid fæces.

Plate V.

R Mintern lith

ON THE

MINUTE ANATOMY OF TWO BREASTS,

THE

AREOLÆ OF WHICH HAD BEEN THE SEAT OF LONG-STANDING ECZEMA.

BY

HENRY TRENTHAM BUTLIN, F.R.C.S.,

SURGICAL REGISTRAR TO ST. BARTHOLOMEW'S HOSPITAL.

IT is probably known by many members of this Society that within the last year or two our president has called attention in a short article in the St. Bartholomew's Hospital Reports [1] to the occasional occurrence of carcinoma of the breast in apparent connection with long-continued chronic eczema about the nipple. This relation has been noticed by other of the surgeons attached to St. Bartholomew's Hospital, several of whom are so deeply impressed by the feeling that the eczema stands to the cancer in the relation of cause to effect, that it has fallen to my lot to examine two breasts which were removed completely on account of the eruption, although there

[1] Vol. x, 1874, p. 87, 'On Disease of the Mammary Areola preceding Cancer,' by Sir James Paget, Bart.

was not at the time of the operation any evidence of cancer.

No. 1.—The first was removed by Mr. Savory in the case of a lady, 57 years old, who had suffered from an extremely obstinate eczema of the nipple and areola of long duration. No relief followed treatment, although amongst other measures the free application of strong nitric acid was employed; and as the disease had existed for so long a period, and was the source of considerable irritation, the whole breast was removed together with an elliptical portion of integument including the nipple. The breast did not exhibit to the sight or touch any induration save that produced in the integument of the nipple and areola and for a short distance below them. The nipple no longer projected. It appeared to be absent. The whole areola was scabbed over.

No. 2.—The second case occurred in a woman, æt. 52, under Mr. Smith's care at St. Bartholomew's Hospital. The disease had existed three years. Within the last two or three months of that period the breast had become hard at the upper part. This breast was removed after it had been examined in consultation in the operating theatre. The condition of the nipple and areola was very similar to that noticed in Mr. Savory's case, but in addition there was a good deal of induration at the upper and inner part of the breast. After removal this induration had much the same appearance as certain forms of cancerous infiltration.

The changes found on microscopical examination were, to a certain extent, precisely similar in both cases. They may be thus described.

(*a.*) The mucous layer of the epidermis of the part affected by eczema had undergone proliferation and was increased in thickness. It was also occasionally traversed by fine fibrous bands, or fibro-cells or spindle-cells.

(*b.*) The corium and subcutaneous tissue were indurated and infiltrated with small round cells (leucocytes).

(*c.*) The galactophorous ducts were widely open or

distended. They were not lined as in the normal condition by cylindrical epithelium, but contained frequently large-masses of epithelium of the squamous or glandular form. Several of the tubes were completely filled with epithelium.

(*d.*) In the immediate vicinity of the ducts the connective tissue was discovered frequently infiltrated with small round cells, just as were the corium and subcutaneous tissue.

(*e.*) The conditions described in *c* and *d* extended into the breast for an inch or more beneath the nipple.

The further changes found in No. 2 existed in the indurated portions of the breast before mentioned.

(*f.*) The acini were much larger than normal, and were filled with epithelium; they were also more widely separated than is the case in health.

(*g.*) The condition of the ducts and of the tissues surrounding them was similar to that described in (*c*) and (*d*).

The appearances above described naturally give rise to much reflection.

The alterations in the mucous layer of the epidermis and the small-cell-infiltration of the cutis, and even of the subcutaneous tissue, are not astonishing. They are just such conditions as those described by Neumann and Biesiadecki[1] in chronic eczema of other parts of the integument.

But the alterations in the ducts and more especially in their epithelial elements are so striking as to call for special attention. Many of them presented on section much the same characters as those observed in the dilated ducts or follicles of the mammary glandular tumours; some of them were so completely filled with epithelium that they looked like the sections of tubes in tubal nephritis; whilst in others again the epithelium growing out in irregular masses from the walls was traversed by occasional rod-like cells or fibres (as in the mucous layer) and

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[1] 'Text-Book of Skin Diseases' (Translated by Pullar). 1871, p. 141.

brought forcibly to one's mind the early stages of pro-
liferating intra-cystic growths.   I have not, however,
succeeded in discovering any further stages of develop-
ment within the ducts.

The depth below the surface to which the small-cell-
infiltration of the connective tissue in the vicinity of the
ducts extended is also much greater than might have
been expected.

This last feature becomes more important when con-
sidered in conjunction with the enlargement and cell-pro-
liferation of the acini of the lobules in the indurated por-
tions of the gland.   These conditions are very similar to
those which I have several times found in the immediate
outskirts of a carcinoma of the breast.   They are de-
scribed too by Waldeyer,[1] in his articles on the develop-
ment of carcinoma.

Although the indurated portions of the breast, No. 2,
so closely resembled in their naked-eye appearances the
characters of certain forms of cancerous infiltration, there
was not any structure in the sections which I could con-
vince myself had reached the condition of actual car-
cinoma.

The changes met with in the most distant parts were
without doubt continuous with those produced in and
beneath the surface by the eczema.   For, although the
continuity of the disease could not be traced in any single
section (from the very nature of such sections), the dis-
ease was found in every section and at all depths below
the surface.

There being no cancer present in these breasts it is
impossible to say that cancer would have formed in either.
The facts before us, however, are not without importance,
since they show that considerable changes are capable of
being induced in the very substance of a more or less
deeply-seated organ apparently by the presence of a very
slight area of disease on the surface.

[1] 'Virchow's Archiv,' bd. 55 (1872), s. 124, "Die Entwickelung der Car-
cinome."

DESCRIPTION OF PLATE VI.

Mr. Butlin on the Minute Anatomy of Eczematous Breasts.

Fig. 1 shows a section cut down through the eczematous nipple into the breast: magnified from 5 to 10 times (4 in. objective). *a.* Small cell-infiltration in cutis and subcutaneous tissue. *b.* Ducts cut across. *c.* Small cell-infiltration in vicinity of ducts. *d.* Scab on surface.

Fig. 2.—Portion at *a*, more highly magnified.

Fig. 3.—Epithelium in duct.

$\frac{1}{1000}$ in. |__|__|__|__|__|__|__|__|

Figs. 4 and 5 show small cell-infiltration in vicinity of ducts.

$\frac{1}{1000}$ in. |__|__|__|__|__|__|__|__|__| oc. 3, obj. 7, tube drawn out.

Fig. 6.—To show characters of epithelium in ducts.

|__|__|__|__|__|__| oc. 3, obj. 7, t. d. o.

Fig. 7.—Section of duct filled with epithelium, less highly magnified.

$\frac{1}{1000}$ in. |__|__|__|__|__|__|__|__|__|__|__|__|__| oc. 3, obj. 4, t. d. o.

Fig. 8.—Section of nodule of indurated breast, magnified as Fig. 1, showing ducts (*b*) and acini in fibrous tissue.

Fig. 9.—Acinus, highly magnified.

$\frac{1}{1000}$ in. |__|__|__|__|__|__|__|__| oc. 3, obj. 7, t. d. o.

Fig. 10.—Diagrammatic sketch of portion of Fig. 8, more highly magnified—duct and acini (A, 1 in.).

Plate VI

FIG I

d

a

c

c

b

d

FIG O

Mintern Bros

CASE

SPHACELUS OF THE THYROID GLAND,

WITH

RECOVERY OF THE PATIENT.

BY THE LATE

GEORGE GREEN GASCOYEN, F.R.C.S.,

SURGEON TO THE LOCK HOSPITAL, AND ASSISTANT SURGEON TO ST. MARY'S
HOSPITAL, ETC.

THE patient was a Frenchman, aged 38, of dark complexion, well nourished, and healthy, though in the habit of drinking freely.

For ten days his neck had been getting stiff, and latterly painful on movement, whilst his throat had become so sore and swollen that he could scarcely swallow. Although feeling very ill he had continued his employment, and had not sought medical advice.

On March 29th, 1873, he consulted my friend Dr. Vintras, who found great swelling of the front of the neck as low as the sternum, and extending laterally beyond the sterno-mastoid muscles. The skin was of a bright red colour, and extremely sensitive to touch, so that examination was difficult; the swelling was dense and

brawny, pitting slightly on pressure about the median
line, but no fluctuation could be detected. The pain and
thickening prevented movement of the head and depres-
sion of the jaw, so that the man was quite unable to
open his mouth or to protrude his tongue. He could not
speak distinctly, and could swallow fluids only with con-
siderable difficulty. He was evidently suffering intensely,
and there was much febrile disturbance; the face was
congested, but there was no interference whatever with
respiration. As he would not enter the hospital he was
ordered to foment and poultice his neck, and to take
liquid nourishment with bark and ammonia : morphia
was also given.

On April 1st, when he next attended, the skin had
given way on the right side of the neck, leaving a small
aperture through which dark offensive pus issued. He
was somewhat relieved by the discharge, but there was
little alteration in the swelling or in his general condition.
The right mamma and cellular tissue adjoining were
acutely inflamed, and threatened to form matter. The
patient was in great pain and could scarcely speak or
swallow, but he refused to have an incision made in the
neck.

He was seen at home on the 4th, being too ill to leave
his bed. The skin in the front of the neck had given
way in two or three places, through which sloughs and a
quantity of foul pus were escaping ; some large pieces of
detached cellular membrane were drawn away, but much
more could be seen through the openings. The swelling
was somewhat less, and he could swallow rather better,
but he was very low and suffering much from irritative
fever. He had taken scarcely any food during the last
three or four days, had slept but little, and was delirious
at night.

I saw him on the following day, the 5th, with Dr.
Vintras. There was then a large irregular opening in
the front of the neck, measuring about three inches
transversely by nearly two inches vertically, with ragged

gangrenous edges; through this a large piece of dead tissue was protruding, and there was a very copious discharge of dark-coloured matter; the stench was intolerable, and the man complained bitterly of it. With a pair of dressing forceps a flattened solid mass was readily pulled away, together with several large sloughs; many other pieces had also come away during the night in the discharge. When these had been removed and the pus sponged out the larynx and trachea were seen lying bare at the bottom of the wound, with the sternal muscles deprived of their sheaths, passing up to their attachments, but there was no trace of the thyroid body. The layers of fascia and the connective tissue had completely disappeared, so that a living dissection of the parts in the front of the neck was exposed. The finger was passed up to the hyoid bone, over the laryngeal cartilages, and along the rings of the trachea, until it reached the upper margin of the sternum. On either side the carotid artery could be felt pulsating at the bottom of the wound, but no portion of the thyroid gland remained. The muscles and other structures were covered with red florid granulations. The man could now talk fairly and protrude the tip of his tongue; he swallowed fluids readily, and the movements of the larynx, though less active than usual, were very distinct. The skin of the neck beyond the sterno-mastoids was discoloured, and there was much swelling, but the inflammation was subsiding. He was nearly free from pain, had slept fairly, but was at times delirious; he had taken liquid food largely, the bowels had been well relieved without medicine; pulse 108, with considerable power, and his aspect was good. The swelling about the mammary gland was fast passing away. A large collection of pus which had gravitated over the first piece of the sternum was evacuated by incision.

On examining the pieces of slough the larger one proved to be the thyroid body which had separated *en masse.* The gland was flaccid and shrunken, but its

outline was perfect, the lateral lobes connected by the isthmus were easily recognised, and their subdivision into small irregular lobules could be seen through the thin fibrous capsule.

From this date the patient made a rapid recovery, and at the end of a week the large wound in the neck was nearly healed; free movement of the head was, however, much impeded by the inflammatory thickening which still remained, but this was gradually regained.

The man has since enjoyed good health, and has experienced no discomfort whatever from the loss of the gland and connective tissue; the act of swallowing is perfect, and the larynx can be felt to move as usual beneath the skin. No adhesion has taken place between the cicatrix and the parts beneath, as might have been feared, and the movements of the lower jaw and neck are performed as quickly and readily as before, although they are perhaps slightly limited in extent. From an examination of the patient at the present moment it is difficult to believe that he has been the subject of such an extensive loss of tissue.

Remarks.—This case is of interest in showing that death of the thyroid gland may follow an attack of idiopathic inflammation in a person apparently healthy, and that no inconvenience is suffered by its loss. It also confirms the opinion expressed by those few writers who have touched on the subject, that this organ shows an extreme indisposition to become inflamed.

Dr. Copland says that "inflammation of the thyroid gland never occurs but in scrofulous habits,"[1] and Sir Thomas Watson[2] endorses this opinion. Little more than a passing mention of inflammation of the thyroid body as a possible contingency is to be found in our standard works; and although a few instances of suppuration in this gland have been recorded, most of them have occurred in cases of bronchocele which had become

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[1] 'Dictionary of Practical Medicine,' vol. i, p. 269.
[2] 'Principles and Practice of Physic,' 4th edit., vol. i, p. 796.

accidentally inflamed.[1] The late Professor Porta, however, in his ' Treatise on Diseases of the Thyroid Gland,'[2] devotes a chapter to thyroiditis, wherein he states that inflammation may commence in the cellular envelope or in the parenchyma of the gland; that it may be limited to one side, or involve the whole organ in acute inflammation; and that when inflamed the gland suddenly increases in size, so that it will become very large in a day or even in an hour. He mentions sphacelus as a very rare and fatal result, of which he has scarcely seen an example.

Lebert[3] in his valuable work on the diseases of this gland bases the section on thyroiditis upon 50 cases, of which 9 occurred in his own practice. Of these 13, or 26 per cent., proved fatal, and in 32 of them, which resulted in the formation of matter, 11, or 34·37 per cent., died. Whilst admitting that goitre is one of the predisposing causes, he states that idiopathic inflammation most frequently occurs in a healthy gland. He alludes to seven examples of mortification of the gland which he has collected from various German authors, and gives the details of two of them which very closely resemble those met with in the subject of this communication.

In these very rare instances of complete necrosis Lebert is of opinion that the inflammation does not take place in the gland itself, but in the connective tissue supporting the thyroid arteries, which, by causing thrombus in the vessels, leads to death of the entire organ. The shrunken, but otherwise unchanged, condition of the gland in the present case and the extensive sloughing of the cellular membrane afford strong testimony to the accuracy of this surmise.

All writers speak of dyspnœa and dysphagia, occa-

[1] Allan Burns, ' Surgical Anatomy of the Head and Neck,' p. 215; ' Holmes's System of Surgery,' vol. iv, p. 706, 1st edit.; ' Gross's System of Surgery,' vol. ii, p. 390, 3rd edit.; ' Chelius's System of Surgery,' by South, vol. ii, p. 653.

[2] ' Delle Malattie e delle Operazioni della Ghiandola Tiroidea,' Milan, 1849.

[3] ' Die Krankheiten der Schilddrüse und ihre Behandlung,' Breslau, 1862.

sioned by pressure of the swollen gland or of matter upon the air-passages and œsophagus, as the most constant and urgent symptoms of acute thyroiditis; and of the 13 fatal cases referred to by Lebert death was caused in 5 by asphyxia from compression, and in 4 by bursting of the abscess into the larynx or trachea. In the case now before us, however, there was no difficulty of breathing, although the face was much congested from venous obstruction; and although the dysphagia was very great it appeared to result from the interference with the movements of deglutition dependent upon inflammatory exudation into the tissues, rather than from pressure upon the œsophagus.

Of the 7 cases of mortification mentioned by Lebert 4 recovered, and in these, as in the present example, no after inconvenience of any kind was observed although the gland was completely destroyed.

The extirpation of this body by natural means constitutes a very interesting physiological experiment, and the absence of any appreciable effect sustained by its loss is in full accord with the experimental excision of this gland which has been occasionally practised upon animals, and with its more or less complete removal by operation in the human subject.

That no apparent disturbance of function should attend the loss of the thyroid body would lead to the inference that in the adult it has become an effete organ, of little, if indeed of any, service in the economy, whatever its value may be in early life during the period of active development; and perhaps of some interest in connection with this latter part of the subject are the two cases of congenital absence of the gland in children, recorded by Mr. Curling in the ʻ Transactions ʼ of this Society,[1] in both of whom " defective cerebral development " was combined with arrest of growth.

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[1] ʻ Med. Chir. Trans.,ʼ vol. xxxiii, p. 303.

NOTES

ON THE

BOUTON DE BISKRA

(MYCOSIS CUTIS CHRONICA, *AUCTORIS*).

BY

H. VANDYKE CARTER, M.D. LOND.,
INDIAN ARMY.

(COMMUNICATED BY J. BURDON SANDERSON, M.D., F.R.S.)

THERE have long been known certain endemic skin-affections which in English are commonly termed " boils " or " sores," in French " boutons " or " clous," in German " Beulen," &c.; other more vague definitions abound, but it will be enough to remark here that the maladies in question are found in countries so wide apart as the west coast of Africa, the West Indies, certain oases of the Sahara, parts of Turkish Arabia, the shores of the Red Sea, Aden, Northern Sind, the Punjaub, around Delhi, and in the North-west Provinces of the Bengal Presidency, &c. Quite recently, also, I saw in the Island of Crete many examples of an endemic " boil " known as the " Caneotica," which is to be regarded as an imported form of the so-called " Aleppo evil."

Of all these peculiar skin-diseases good descriptions exist in medical works, but with very rare exceptions, so far as I am aware, nothing definite has been elicited as to their real nature and structural character; and it was with the hope of acquiring some much-needed information that in October last (1874) I visited the oasis of Biskra in Algeria, which is situated on the border of the great desert of Sahara.

The object of my search—well enough known as the " Bouton de Biskra "—has been compared with the Delhi boil and the Aleppo evil (*vide* Nos. 822 and 823 at p. 173 of the ' Nomenclature of Diseases,' London, 1869), and since these two last-named affections are familiar to medical officers in India as very troublesome complaints, whose true character it would be useful to ascertain, I obtained the generous permission of H.M.'s Secretary of State for India in Council to visit Algeria (amongst other localities) on my return to duty in the Bombay Presidency. Notwithstanding previous intimation, the month of October proved to be too early in the season, and I saw only a few cases of the " clou;" but this deficiency has been largely supplemented by the persevering exertions of my friend Dr. E. Weber, Médecin-Major, 3me Batn. d'Afrique, who furnished me with new information on points I had arranged to inquire into, and who has most kindly supplied me with the " pièces anatomiques " forming the subject of these notes. Other details and drawings which I had collected have been already forwarded to the India Office, and I do not propose in this place to enter upon a complete account of the bouton de Biskra, as I hope for future opportunity of discussing the entire class-of affections to which it may be said to belong.[1]

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[1] It may, however, be useful to add that the complaint in question commences as a superficial papule or pimple, which in the course of some days becomes a nodule or " bouton," and then acquiring a scab, often ends as a very indolent ulcer. The ordinary duration of the eruption is five or six months, or from November to April, and the spots may be numerous, secondary ones appearing round the first or at a distance. Very little local

The specimens from Algeria reached Bombay in June last; they had been placed in a solution of potassic bichromate, and though rather compressed were in good state of preservation, being of normal consistence throughout and free from any sign of decomposition. Respecting them Dr. Weber writes as follows:

No. 1. "Les morceaux sont des clous de Biskra au début; ils ont débuté chez le nommé Eurquet, soldat au 3e Batn. d'Afrique, mort le 5 Janvier, 1875, de phthisie pulmonaire; ces clous avaient 15 jours d'existence lors de la mort du sujet: chaque portion du clou excisée est entourée d'un morceau de peau saine; ils ont été pris sur les bras."

No. 2.—"Les morceaux viennent du nommé Rebours, soldat au Bataillon d'Afrique, mort le 16 Janvier 1875. L'un de ces clous etait ulcéré. Tous sont entourés d'une partie de peau saine: cet homme est mort d'une albuminurie (Maladie de Bright). Les clous ont été pris deux sur les bras, l'autre sur le front."

There were also specimens of a cicatrix of ten years' standing, and a bit of healthy skin sent for purposes of comparison.

I now proceed to describe the minute structure of these preparations, and will afterwards add a few remarks on the facts elicited.

No. 1. Early stage of the "clou." Three specimens. After their long immersion in the bichromate little change was to be seen by the unaided eye, and upon section only a bulging of the surface with a slight brownish infiltration of the cutis, which indicated the situation of the

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pain or redness attends the "clou," and as a rule there is no constitutional disturbance whatever. Afterwards a scar is left having a punched-out character and little disposed to contract; it long retains a brownish tint and is almost indelible. The ulceration when extensive assumes a serpiginous character. The commoner sites of the clou are the face, arms, and legs.

Unless the Anglo-Saxon word "boil" means both "tuber" and "ulcus" its application to the present instance is unsuitable. There is, perhaps, nothing of a furuncular character in the clou de Biskra, and this complaint has no connection with syphilis, &c,

bouton. In different instances this measured $\frac{1}{8}$ to $\frac{1}{3}$ inch long and rather less in depth, and though without any definite form was yet tolerably defined at its boundaries. After hardening in alcohol vertical and transverse sections of the "bouton" were made, and the thin slices were then placed in glycerine, to which acetic acid, liquor potassæ, and carmine solution were separately added. No other reagents than these were employed. Both high and low microscopic powers were used, the light being good and the air warm and dry or moist. There appeared no reason whatever to attribute the incidence of the parasitic growth described below to accidental causes, and such moulds as are apt to spring up in glycerine preparations present a totally different aspect.

After close scrutiny with the microscope I conclude that the clou de Biskra is essentially a granulation tumour, $i.\ e.$ a tumefaction caused in chief part by the advent of pale, round cells measuring $\frac{1}{4000}$ to $\frac{1}{2000}$ inch in diameter, and which, becoming densely crowded in the cutis, produce expansion of the connective-tissue meshes, effacement of the papillæ and the disappearance of the adjoining softer epidermis. Compression of the hair-sacs, with extrusion of the hairs and envelopment of the sweat-ducts, are noticed; and following the course of these ducts collections of similar cells are found, extending into the subcutaneous areolar tissue, and there accumulating around the sweat-glands themselves. Blood-vessels are numerous and enlarged, especially in the corium; nerves and fasciculi of smooth muscular fibre are to be seen, often also surrounded by the pale cells; but none of these structures of the skin, whether extrinsic or intrinsic, are essentially altered in appearance. For illustration of these remarks see Plate VII, figs. 1 and 2.

It would certainly seem that this cellular infiltration commences in the more superficial part of the cutis; and there, too, the parasitic growth I am about to mention first makes its appearance. The cell production is always well-defined in its limits, and would seem to be

superadded to the tissues rather than derived from them. So far nothing very peculiar was made out, since several skin tumours are mainly composed of these pale cells, which are variously termed lymph, granulation, migratory or embryonic, &c., corpuscles, but in searching further I at length detected the appearances depicted in Plates VIII and IX, fig. 1. Thus, permeating the cellular infiltration, and especially frequent at its out-skirts, were seen numerous bulging and branching chan-nels, which were usually filled with filamentous or myce-lioid structures. On transverse section, round spaces appeared similarly occupied, and it became evident that the lymphatic vessels of the parts implicated were the seat of a foreign growth, which can only be compared with that which is characteristic of the lower vegetable organisms. Generally the walls of these channels are thin, but in the papillary portion of the cutis a condensation of tissue due to expansion of the local lymph canal may be seen around them; an endothelial lining may often be detected, and the general arrangement of the canals in question leaves little doubt of their being lymphatics in various degrees of distension. Generally their diameter attains the $\frac{1}{500}$ to $\frac{1}{300}$ inch, and sometimes they are so wide as to merit the name of lacunar spaces.

It is, however, in their contents that interest chiefly centres. Lymph-cells are not seen, and seldom anything like coagulated plasma, but there is instead a form of growth which must, I think, be regarded as strictly parasitic in its characters. The occurrence of granular micrococcus-like masses in these lymphatics would have been striking enough, but to find so clear proof of mycelium structure was wholly unanticipated. The con-junction with this of spheroid and zoogloea forms, in the same localities, removes all obscurity as to the character of the new organisms.

The mycelium structure is composed of bright fila-ments measuring $\frac{1}{20000}$ to $\frac{1}{15000}$ inch in breadth, and seldom more than $\frac{1}{800}$ inch in length; they are usually straight or

slightly wavy, branch and divide dichotomously, and
commonly at open or even right angles, so that the
collection has the crooked aspect of a bundle of thorns or
an intercrossing of spider's webs. The interlacing of
fibrils is therefore not very close, and this open aspect
together with the " bent " look of the whole might be
worthy of a *special* distinction.

Sometimes alone, but commonly intermixed, are found
bright, refractile spherules, and it occasionally appears
that the filaments may be constructed of rows of these
particles; but more often it is evident that the terminal
branchlets of the mycelium furnish, by a budding process,
one or more of the bright granules which should hence be
compared with spores or conidiæ. Probably these spores
also undergo germination on the spot of their production,
and, in fine, it has seemed to me as if the whole process of
development were as follows :—Free micrococci grow into
masses in which a mycelium appears, and from this arise
spores which, by accumulation, produce an aspect again
approaching the original zoogloea-form, only that the
last-formed individual particles are larger than the first
were. What next occurs will be afterwards surmised,
and several details omitted in this brief narrative may be
noticed in the Plates VIII and IX hereto appended.

Neither acetic acid nor solution of potass destroy the
growths. The latter are almost colourless, and only in
the mass do the micrococcus groups assume a brownish
aspect. I am unable to say whether or not the mycelium
filaments are jointed; sometimes they have a beaded
contour, but even this is not always apparent, the edges
seeming parallel; in general, the differences noticed in
either filament or granule, or in the relative proportion of
the two, are inconsiderable and readily explicable by
reference to varying stages of growth.

Nowhere else than in the one situation named have
these appearances been detected; and I would add that
when they become familiar to the observer, he may after-
wards find them in parts of the tumour where at first they

were passed over. A good light, high powers, and careful focusing have enabled me to see this remarkable parasitic growth in almost every part of the "clou," but where the cellular infiltration is unusually copious there it may be impossible to find it. The growing part is evidently the margin of the tumour, and principally that edge close to the surface of the skin. At the superficies, too, the clou seems to commence, and I have traced a distended lymph-tract so close to the free surface that it seemed as if by an irregular channel through the epidermis itself, its contents might be extruded or foreign matters enter inwards.

Respecting the source of the pale, round, infiltrating cells, I would only remark that although they were most abundant around the distended blood-vessels, yet they were numerous elsewhere; and such of the like as may have been contained in the lymph channels were very effectually driven away, since none could be seen in the midst of the advancing parasitic growth. Possibly this expulsion would partly account for the cellular infiltration which attends the progress of the implanted organisms, and certainly the whole formation of the "bouton" is a slow and gradual process.

Specimen No. 2. — Late stages of the bouton de Biskra. Under this head are included an ulcerated "clou" from the forehead, and two others from the arm; actual duration of disease not mentioned, but known to be advanced. Sections were made and treated as in No. 1.

The pieces taken from the arm presented a general aspect like that above described, only the reddish-brown infiltration was more evident to the eye and of softer consistence. The surface was also more irregular, and the still persistent cuticle had here and there been pushed outwards by force from within, with the result of leaving depressions which had the aspect of follicular involutions of the epidermis, but no morbid element was noticed in connection with these alterations of the cuticle,

even when they were considerable. On minute examina-
tion the bulk of the tumour was found to be still
granulation-tissue; no marks of fatty degeneration in the
cells were present; blood-vessels abounded. Groups of
pale cells were seen deep down in the subjacent connective
tissue, but the more striking feature here, and to a
rather less extent superficially, was the abundance of
bright, orange-coloured particles, almost everywhere
scattered throughout the tissues, and specially numerous
around the sweat-glands, ducts, and larger vessels (see
Plate VII, figs. 3 and 4). Commonly grouped in small
clusters, their individual size varies from $\frac{1}{10000}$ to
$\frac{1}{2500}$ inch; their form is rounded or rarely ovoid, or
dumb-bell shaped as if dividing. They resist the action
of acetic acid and solution of potash.

Several varieties in their aggregation were visible, but
as many groups were in form regular, being ovoid or
elongated, and as much as the $\frac{1}{800}$ inch long, with a clear
contour and evident organic connection, it must be al-
lowed that some uniform influence determines their form
and position, and I infer that these remarkable bodies are
properly located in the interspaces of the larger bundles
of connective tissue. · Certainly some of the orange-
tinted masses may be contained in lymph-spaces lined by
endothelium, for in addition to an evident linear arrange-
ment, I have noticed that the endothelial cells partake of
their hue, becoming studded with tinted granules; and
since the finer lymph or serous channels are everywhere
widely distributed, the complete dispersion of these
coloured bodies becomes easily explicable. That the
coloured particles originate within the granulation-cells is
not likely; and even the more plausible idea that they are
derived from disintegrating red blood-globules is, not-
withstanding the rare occurrence of free hæmatoidin-like
· masses, by no means well founded. Appearances have at
times suggested that the ovoid and more capsulated
groups of particles (which might be compared to *thecæ*)
are borne at the end of mycelium filaments of large size,

which exist in the tissues themselves; yet on re-examination of the specimens it has always been doubtful whether the abounding elastic fibres had not simulated such mycelioid growth; and, in short, I am unable to account for the presence and dispersion of these characteristic bodies, except on the supposition that they represent a further—perhaps terminal or fructificational—stage in the growth of the undoubted parasite which has been shown to be present in the early period of the " clou." If this view be correct, the occurrence of such remarkable products is a fact both novel and interesting.

In these later specimens of the " clou " the true mycelium growth was not seen, but occasionally micrococcoid masses were found, which were sometimes shrunken or withered in aspect, and at other places were in seeming connection with the tinted groups as indicated in Plate IX, fig. 2. From this observation there is derived confirmation of the opinion that a final stage of the entire parasitic growth is represented by these coloured bodies.

The specimen of ulcerated " clou " from the forehead is noteworthy from its greater vascularity, and the abundance of round and stellate granulation-cells; and the large papillary eminences seen on its surface would imply that a sort of " fungating " sore had existed. The ulcer was well-defined and its dessicated secretion was abundant; but no peculiar element in this discharge could be detected in this specimen. In no part were vegetable organisms to be seen, and the orange-tinted particles were few in number; hence one might suppose that the parasitic growth, with its products, had become mostly expelled, and such is a probable consequence in all cases of active or prolonged ulceration of the clou.

Dr. Weber has remarked that the open-ulcer form heals more quickly than the non-discharging squamous or scabbed variety of this affection.

I found fat-cells and striated muscular fibre at the base of the ulcer in question, but in localities of the body where the subcutaneous connective tissue is more abun-

dant than on the forehead, so deep a penetration of the
sore as is here implied is rarely seen. The sweat-glands
were noted as particularly large, their contents were un-
changed in aspect.

The decided reddish hue of the substance of these
'boutons' is doubtless partly due to the presence of the
small orange-tinted masses above described.

Specimen No. 3.—An old cicatrix taken from the fore-
arm of an Arab, who died in consequence of fracture of
the skull. The scar was said to be of ten years'
duration. Repair was almost complete; the cuticle was
smooth and thin, pigment abundant, papillæ few and
irregular; loose connective tissue was found in place of
the corium, and a few hairs and sweat-glands had
survived. In several places close to the skin I noticed a
scanty collection of the red masses, mostly disposed in
thin streaks; nothing else abnormal was detected. Hence
one would infer that on the formation of a scar hardly
anything remains of the original "clou."

The portion of healthy skin which I examined presented
no trace whatever of change; it had been treated in
precisely the same manner as the pathological specimens.

Remarks.—The most obvious fact which has been
elicited in the preceding observations is the close and
constant association of a parasitic growth with the
bouton de Biskra; and the question now arises whether
or not the former be the essential cause of the latter.

In the absence of synthetic experiment (which was
clearly impossible in the present instance) this question
cannot strictly be answered, yet analogy and several
collateral circumstances would point to affirmative reply.

Undoubtedly the presence in the tissues of a foreign
growth would be a sufficient reason for all the signs of
irritation which are seen in this tumour of the skin; and
the following features of the complaint are adequately
explained on the affirmative decision.

The " bouton de Biskra " is, like the " Delhi boil " and
" Aleppo evil," (*a*) an endemic affection; it is (*b*) seasonal

in its occurrence, has an incubation period, and is limited in duration; (c) the site of the clou, or the first clou, is usually on parts exposed to contamination; (d) the spots are commonly multiple and seldom recur. Here, I would remark, is evidence of a constitutional nature singularly analogous to the phenomena of "variola;" and it would not be unreasonable to infer that it is through the lymphatic vessels that the system becomes affected.[1] Something may be added by the vegetable organisms I have described, which exerts a protective influence; and the successive cropping of the local manifestations may be regarded as other evidence of local and systemic infection.

(e) Although the clou de Biskra is not to all appearance a contagious malady, yet there is evidence that it may be propagated by inoculation. Most, if not all, residents at the station some time or other acquire the disease, and persons are affected even after leaving the locality.[2]

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[1] Having before considered this point I begged Dr. Weber to inquire into it, and he writes as follows :—" Biskra, 23rd April, 1875.—Cette année-ci les clous ont été très nombreux, et guidé par vos avis et vos conseils, j'ai trouvé dans la plupart des cas un retentissement vers les ganglions lymphatiques, se propogeant à ces ganglions par un chapelet de petits ganglions engorgés le long des vaisseaux lymphatiques: je vous ai même envoyé le clou d'un homme mort à la suite d'un érysipèle de la jambe qu'un clou au pied avait déterminé (this specimen did not reach me.—H. V. C.); depuis, j'ai vu plusieurs cas du même genre, qui n'ont pas eu une terminaison aussi funeste, mais qui ont donné lieu à des abcès le long des lymphatiques. En ce moment même j'ai encore en traitement à l'hôpital un homme qui a un clou au pied avec un érysipèle de toute la jambe jusqu'au genou, et du genou part un chapelet de ganglions qui va répandre aux ganglions de l'aine: vous voyez par celà qu'en général les glandes lymphatiques ne sont pas épargnées, quoique d'habitude les complications de ce côté sont peu graves."

[2] It was, of course, important to determine the question of inoculation, and Dr. Weber writes of his proceedings thus:—" Des personnes arrivées à Biskra en automne ont eu des clous 7 jours après leur arrivée, et dernièrement un Vétérinaire qui est parti pour la France a eu des clous 30 jours après son départ de Biskra. Les inoculations que j'ai faites à Biskra et que j'ai faites faire à Philippeville avec le liquide du clou n'ont pas donné de résultat concluant, mais m'étant conformé à votre conseil d'inoculer les croûtes, je suis arrivé à un beau résultat. Voici le fait. Le 7 Avril 1875 tous les clous étant presque guéris, j'ai pris un homme, soldat au Batn. d'Afrique, âgé de 25

I may here remark that the Delhi boil and Alèppo evil are known to be inoculable, and in Crete I was informed that the Caneotica is certainly even contagious. The same property attaches to the frambœsia of Africa and the W. Indies, and if the observations recorded in the text of these "notes" are applicable to the maladies just named, no inconsiderable light in this respect is, as it seems to me, hereby thrown upon the whole series of them.

I do not propose pursuing this topic, and will only add that until now no efficient cause of the malady in question has been made known. I have carefully perused most of the original essays of French authors, and cannot but conclude that none of the assigned meteorologic, telluric, or dietetic influences usually invoked can be regarded as the *vera causa* of the bouton de Biskra.[1] An inkling of the probable truth was, however, long since

ans, ayant un clou de trois mois à l'avant-bras ; n'ayant aucun antécédent syphilitique et d'un bon tempérament. J'ai pris un morceau de la croûte, je l'ai réduit en poussière et j'ai inoculé sous l'épiderme quelques graius de cette poussière à 3 soldats et à M. Maty, mon médecin aide-major ; les trois soldats ayant un peu de crainte l'inoculation n'a pu être faite dans de bonnes conditions, mais chez le docteur Maty, agé de 27 ans, à Biskra depuis 8 mois, n'ayant aucun antécédent syphilitique, j'ai bien introduit la poussiere de la crônte sous l'épiderme de la cuisse droite. Trois jours après, la piqure prenait le caractère du clou de Biskra au début, et aujourd' hui 23 Avril, ce clou à forme ulcéreuse, ayant le fond de la plaie fougeux, est grand comme une pièce de 2 sous, c'est-à dire qu'il a trois centimêtres de diamêtre, et n'ayant aucune tendance à la guerison ; il n'est pas enflammé ni douloureux ; cependant dans les premiers jours les glandes lymphatiques de l'aine ont été un peu engorgées, sans douleur mais aujourd'hui cet engorgement a disparu. Voila donc un clou inoculé ayant maintenant 15 jours, avec tous les caractères du clou de Biskra, que j'ai fait constater par tous les médecins presents ici et par d'autres personnes qui ont l'habitude de voir de ces clous : et de plus, depuis quelque jours trois autres petits clous sont en train de se développer sur la même cuisse à environ 15 centimêtres audessous du premier qui est assez grand." Dr. Weber continues, " toutes ces raisons me fait croire que le clou de Biskra est une affection spécifique dont la cause m'est inconnue."

[1] It need not be denied that the system may be infected by a parasite otherwise than through the channel of the skin ; and in such case, as doubtless holds good for some outbreaks of the clou subsequent to the first, the cutaneous eruptions would be of a "secondary" character.

felt by Virchow, who sagaciously remarks ('Pathol. of Tumors,' Fr. trans., vol. 2, p. 526) of the bouton d'Alep, de Biskara, Sind and Delhi—"The course of these affections is so peculiar that one would be almost tempted to attribute the disease to the presence of some parasite; but there are no data on this point." Subsequently, indeed, medical officers in India have made valuable observations on the Delhi boil, but no undoubted parasitic growth, as I think, has been established to account for the complaint; and it was mainly by way of preparation for further research in this country that I undertook the visit which has, after some interval, proved to be thus not unproductive, and which may by the results now recorded stimulate other observers to inquiries not less promising than was that touching the bouton de Biskra.

Addendum.—Constitutional treatment has not been found to influence the course of this affection; and, locally, caustic applications are alone of any use. Change of residence is beneficial.

Depending on the accuracy of the view that the bouton de Biskra is caused by a lowly organised parasitic growth of plant-nature, I have ventured on the name *mycosis cutis,* adding the word *chronica* to indicate a distinction from the similar acute specific diseases. Perhaps, by-and-bye, this proposed designation will prove suitable for the whole class of these endemic skin-affections, including the Aleppo evil, Delhi boil, framboesia, and the like.

As regards the place of the specific-invading organism above described I would not yet attempt its identification, for this would be premature.

PLATE VII.

Mycosis cutis chronica : vertical sections of the Bouton de Biskra.

Fig. 1.—Vertical section of a clou de Biskra at the early stage (duration fifteen days): × 60 diameters. The shaded tint indicates the general disposition of the cellular infiltration which forms the mass of the tumour and surrounds the blood-vessels, ducts, &c., being prolonged with these into the connective tissue subjacent to the skin. Its chief seat is in the corium, and by its accumulation there the papillæ are effaced and a bulging, yet unbroken contour is produced.

Fig. 2.—A portion of the above magnified 300 diameters. It is taken from near the free surface, and shows the so-called " granulation-tissue " of which the bulk of the " bouton " is formed. On the right, the orifice of a distended blood-vessel is depicted.

Fig. 3.—Vertical section of an ulcerated " clou " made near to one edge : × 40 diameters. To the left are seen a few unchanged or little enlarged papillæ : the granulation-tissue is abundant and forms the basis of the ulcer, being also prolonged downwards. At * is the jagged surface of the ulcer and irregular masses of epithelium, &c., are seen projecting from it. The sweat-glands (one is indicated at †); their ducts and some blood-vessels are seen to be surrounded by dark granular streaks, which represent the orange-tinted bodies described below and in the text.

Fig. 4.—A portion of the above magnified 300 diameters : acetic acid has been added. It shows the form and arrangement of the orange-coloured granules and masses found at this late stage of the " bouton :" their bright appearance is represented in black tint. To the left is part of a sweat-gland; to the right a bit of granulation tissue.

PLATE VIII.

Mycosis cutis chronica.—The parasitic growth in the lymphatic canals, vertical section.

Fig. 1.—Vertical section of the deeper part of a " bouton " at the early stage : × 500 diameters : with acetic acid and glycerine. A sweat-duct passes down the centre; and on its right, below, is a dilated lymphatic canal containing the parasitic growth, whose interlacing filaments are represented in its interior. To the left, above, is another lymphatic trunk filled with the growth, and the two canals unite at the top, where a large-grained micrococcus-mass is seen projecting in the lumen of their common trunk : around here are numerous pale cells, but elsewhere the connective tissue is yet

Plate VII

FIG 1

FIG 3

FIG 2

FIG 4

Plate VIII

Med Chir Trans Vol LIX

FIG I

Plate X   Med Chir Tran, Vol LX

Fig

free of infiltration. Both mycelium-filaments and spherules (conidia ?) are to be seen within the lymph-lacunæ: and in other specimens more minute spheroids were as well observed.

Fig. 2.—Shows a few of the filaments and spheroids in a part of the above: × 700 diameters. A beaded or jointed aspect of the former is perceptible, and the origin of the latter by pullulation at their tips seems to be indicated.

Fig. 3.—Mycelium-filaments from another collection: × 700 diameters: treated with acetic acid. As a variety, they are smoother and less angular than is commonly seen.

PLATE IX.

Mycosis cutis chronica.

Fig. 1.—Transverse section of the edge of a "bouton" in the early stage: in glycerine and acetic acid. × 500 diameters. It shows on the right a divided papilla and the free surface; above are a few granulation-cells; elsewhere are seen the branching lymphatic channels which are occupied by an invading fungus growth. One of these canals is seen issuing from the interior of the papilla, and in this situation dilated cavities are not uncommon; they lodge the parasitic mass, but in thin sections, the latter falling out, they may appear to be empty.

Fig. 2.—Transverse section of a " clou " in the later stage; made at the deeper surface. Treated with acetic acid. × 500 diameters. It shows a dilated channel here making a bend, in which (embolus-like) is contained a granular mass having all the aspect of micrococcoid composition, except that the individual spheroids are of larger size than usual: a detached fragment is seen to the left. Above, the lymph-channel appears to be in communication with some collections of the orange-tinted particles. Other coloured groups are visible to the right, where they are lodged between fasciculi of fibrous tissue: a few larger ones are free. Numerous granulation-cells are also present, so that excepting the mycelium structure (which probably preceded the micrococcoid masses) all the characteristic elements of a bouton de Biskra are here visible.

INTERMITTENT HÆMORRHAGE FROM MALARIAL INFLUENCE.

BY

SURGEON-MAJOR J. H. PORTER,

ASSISTANT PROFESSOR OF MILITARY SURGERY, ARMY MEDICAL SCHOOL, NETLEY.

(COMMUNICATED BY HOWARD MARSH, F.R.C.S.)

HÆMORRHAGE under many denominations has been de-
scribed by surgical writers, but as far as I am aware, and
I have taken some trouble in examining various works
and made inquiry from those likely to have experience in
the matter, periodic hæmorrhage occurring in a surgical
case from an open wound apparently due to malarial
influence has not been described. Such description may,
however, have escaped my notice.

Bearing on the subject Dr. J. Wickham Legg has recorded
several cases of paroxysmal hæmaturia, resembling a fit
of ague, in his valuable essay in the 'Reports of St.
Bartholomew's Hospital' for 1874; and in the 'Medical
Times and Gazette' of the 16th of October, 1875, Dr.
Francis, late of the Indian Army, gives the case of mala-
rial hæmaturia in a child.

It is scarcely necessary to observe upon the fact that

a person having had intermittent fever is liable even many years afterwards to a return of the disease on the occurrence of shock, loss of blood, or constitutional derangement. Sir James Paget, in observing on this fact, has remarked in his ' Clinical Lectures and Essays,' " I have so often noticed this that, whenever I hear of severe rigors following any operation, I ask for a previous history of ague, and have sometimes found that the patient has almost forgotten it in the long lapse of time since he suffered from it." At Netley Hospital this circumstance is frequently observed among the invalids from India. To whatever, therefore, may be the cause which has induced the ague in these patients, to that same cause I attribute the intermittent hæmorrhage in the case I am about to detail; let it be "malaria," "electricity," or "chill," according to the different views which have lately been put forth on the subject, which had become latent.

The subject of the hæmorrhage was a young soldier in the 70th regiment, twenty-two years of age, service five years, sixteen months of which were spent at home and three years and eight months in India. No history of syphilis or hereditary hæmorrhagic diathesis. He appears to have enjoyed good health in India until September, 1873, when at Rawel Pindee he was attacked with intermittent fever of severe form, from which he suffered for upwards of three months, and which suddenly culminated in an attack of rheumatism of both knee-joints. The right knee recovered, but the left continued painful; his health became much impaired, strumous abscesses formed in his neck, and he lost part of the hard and soft palate from necrosis and ulceration. He was sent to the Sanitarium at Murree in April, 1874, but not having benefited by the change of climate was invalided to England in March, 1875.

On arrival at Netley, April 30th, he was in a weak anæmic condition; his weight was only six stone. The left knee was swollen and painful, but, there being no immediate necessity for surgical interference, an endeavour

was made to improve his health, which for some time succeeded, but in July the joint having become very painful, with loss of rest, profuse perspirations, and diarrhœa, it was considered desirable to remove the limb at the knee-joint.

On the 8th of July, by the bloodless method, the patient being under the influence of ether, I amputated the limb by Carden's method, sawing the bone through the condyles of the femur. The operation was followed by the greatest relief; his temperature fell, night perspirations and diarrhœa ceased, and appetite improved.

Matters progressed favorably till the 16th of July, or eight days after the operation, when in the afternoon the patient became chilly and very irritable, stump swollen, painful, and throbbing, followed by flushing of the face, dry tongue, heat of skin, and increase of temperature, with a quick pulse. These symptoms steadily became worse till 5 a.m. on the 17th, when hæmorrhage set in in the form of oozing of venous blood from under the edge of the flap; this continued at intervals notwithstanding elevation of stump, application of cold, and pressure. till 10 a.m., when it was found necessary to place him under the influence of ether, break open the adhesions, and turn out coagula, with a view to discovering the source of the bleeding. No bleeding vessel could be found, the hæmorrhage coming from the surface of the stump and especially from the cancellated bone structure, as if it were forced out of it by pressure from behind.

The surface of the stump was freely exposed to the air, ice-bags placed in the immediate neighbourhood, but until a strong solution of perchloride of iron was applied the bleeding continued. The flap was now replaced, stump elevated, and all constrictions removed, after which, with the aid of 10 minims of tincture of opium, he passed into a quiet sleep, from which he awoke cool, pulse quiet, and no pain or throbbing in the stump; he was, however, very weak, and required a liberal amount of nourishment.

18th,—Report states, patient passed a good night and

is improved this morning. Ordered 10 minims of tincture of ergot three times a day with cool acid drinks.

19th.—At 9.30 last evening preceded by the same symptoms as occurred on the 17th. Oozing from the stump again appeared, when it was found necessary to open it up and apply the solution of iron, which with the application of ice and elevation of limb the bleeding ceased, and he passed into a quiet sleep from which he awoke refreshed.

20th.—No bleeding yesterday; but at 9 a.m. this morning, preceded by the symptoms as already described, oozing again set in, which continued so persistently notwithstanding active treatment, local and constitutionally, that it was feared the patient might sink from exhaustion; he was, however, kept up by the judicious administration of nourishment and stimulants, and he again passed into a quiet sleep, the hæmorrhage stopping.

21st.—At 5 a.m., preceded by the usual symptoms, oozing from the stump again appeared, when the solution of iron was applied, and dilute sulphuric acid and opium administered, but rejected by the stomach; the hæmorrhage, however, ceased as before.

22nd.—Patient passed a good night, but is very weak; the stump has a very unfavorable appearance, presenting to view a mass of black crusts of iron and blood, with a hæmorrhagic smell from it, and no attempt at suppuration.

23rd.—Symptoms of approaching hæmorrhage returned last evening, and notwithstanding the administration of ergot, acids, astringents, and opium, with elevation of stump and application of pressure, oozing set in at 12.30 a.m. this morning, and gradually increased until it was found necessary at 9 a.m. to apply styptics.

The condition of the man having now become alarming, being pale, almost exhausted and pulseless, and the hæmorrhage having presented the characters of periodicity, and bearing in mind that he had suffered from severe intermittent fever in 1873, I decided in consulta-

tion with Professor Longmore to try the effects of quinine, which I added in five-grain doses to the mixture of sulphuric acid and opium, but which his stomach at once rejected. The quinine was therefore given in a simple form with acid, 5 grains three times a day.

24th.—No hæmorrhage since yesterday morning; has taken 15 grains of quinine without any inconvenience. Appearance decidedly improved.

25th.—At 6 p.m. yesterday symptoms of approaching hæmorrhage again set in; temperature rose to 101·7°, pulse 126, but at 9 p.m. instead of oozing from the stump he broke into a copious perspiration, after which he cooled down and passed into a natural sleep. From this time he steadily improved, but not without occasional recurrence of symptoms of approaching hæmorrhage, which having gone through its usual stages ended by action of the skin instead of oozing from the stump.

The quinine was continued, the stump soon threw off its blackened cap, healthy granulations sprang up and he rapidly recovered, adding two and a half stone to his weight. The blood from the stump during a paroxysm of hæmorrhage was carefully examined under the microscope, but nothing abnormal was discovered.

The patient's urine was rather dark coloured, specific gravity 1017, non-albuminous, alkaline; sediment calcareous, containing triple phosphates in abundance.

The attached temperature table will assist in demonstrating the periodic attacks of hæmorrhage:

Case of W. W—, 70th Regiment, æt. 22; service five years; amputation through left knee-joint; recovery.

1875.	Pulse. a.m.	Pulse. p.m.	Temperature. a.m.	Temperature. p.m.
July 2	—	102	—	100·6°
„ 3	96	110	97·8	100·6
„ 4	94	100	99·2	100·2
„ 5	94	114	98·4	101·
„ 6	96	104	98·4	100·8

1875.	Pulse a.m.	p.m.	Temperature. a.m.	p.m.	
July 7	. 92	... 106	... 98·	... 100·	
„ 8	. 95	... 106	... 98·2	... 98·8	Amputation at 10·30 a.m.
„ 9	. 106	... 110	... 98·4	... 98·9	
„ 10	. 107	... 114	... 98·6	... 99·	
„ 11	. 102	... 116	... 98·6	... 99·8	
„ 12	. 103	... 107	... 98·8	... 99·6	
„ 13	. 105	... 116	... 98·	... 100·	
„ 14	. 94	... 105	... 98·	... 99·2	
„ 15	. 97	... 106	... 98·2	... 98·8	
„ 16	. 98	... 114	... 98·2	... 101·6	
„ 17	. 110	... 106	... 98·4	... 98·2	At 5 a.m. hæmorrhage in the form of oozing.
„ 18	. 117	... 116	... 99·2	... 100·4	Hæmorrhage at 9.30 p.m.
„ 19	. 118	... 124	... 99·2	... 101·4	
„ 20	. —	... 110	... —	... 101·2	Patient irritable, but no observation was taken; hæmorrhage at 9 a.m.
„ 21	. 110	... 106	... 98·2	... 98·8	Hæmorrhage at 5.10 a.m.
„ 22	. 102	... 122	... 98·2	... 101·8	
„ 23	. 110	... 130	... 100·	... 100·	Intermittent hæmorrhage from 12.30 a.m. to 9.30 a.m.
„ 24	. 116	... 126	... 98·4	... 101·6	
„ 25	. 107	... 110	... 98·8	... 99·8	
„ 26	. 102	... 108	... 98·4	... 99·8	
„ 27	. 102	... 106	... 98·	... 99·4	
„ 28	. 105	... 110	... 98·2	... 100·6	
„ 29	. 106	... 108	... 98·	... 98·8	
„ 30	. 99	... 102	... 98·2	... 98·6	
„ 31	. 94	... 103	... 98·	... 98·8	

ON THE

ESTIMATION OF ALBUMEN IN URINE BY A NEW METHOD.

ADAPTED FOR CLINICAL PURPOSES.

BY

WILLIAM ROBERTS, M.D.,

PHYSICIAN TO THE MANCHESTER ROYAL INFIRMARY.

(COMMUNICATED BY WILSON FOX, M.D., F.R.S.)

WHEN an albuminous urine is progressively diluted with water and tested from time to time with nitric acid the opacity induced by the acid becomes gradually fainter and fainter until at length it ceases to be visible. This point is reached when the diluted urine contains less than about 0·0014 per cent. of albumen. The more albumen the urine contains the more dilution, of course, it will require to reach the vanishing point of the reaction; and if we could fix this point with accuracy we should arrive at a simple method of estimating the quantity of albumen in urine. The urine could be diluted with water until it ceased to react with nitric acid, and the degree of dilution required to reach this point would be a measure of the proportion of albumen in the urine.

But it is not possible to fix the vanishing point of this reaction with accuracy. It fades away so gradually with increasing additions of water that it is quite impracticable to decide, within many degrees, the point at which it ceases to be appreciable.;[1] and not only so, but the development of the reaction is more and more retarded as the dilution proceeds, until at length it only becomes visible after the lapse of several minutes after the acid is added.

The following experiment on a moderately albuminous urine illustrates the behaviour of albuminous solutions when tested with nitric acid under progressive dilutions with water.

Degree of dilution with water.	Time after the addition of the acid when the reaction comes into sight.
20 volumes, and all lesser dilutions.	Immediately.
30 volumes.	About 4 seconds.
40 „	„ 8 „
60 „	„ 10 „
90 „	„ 20 „
110 „	„ 30 „
120 „	„ 38 „
140 „	„ 50 „
160 „	„ 1 minute.
180 „	„ 1¼ „ faint.
200 „	„ 1½ „ very faint.
240 „	„ 3 minutes, very faint.
280 „	„ 6 „ doubtful ; very faintly visible in 10 minutes.
300 „	No appreciable reaction.

Finding it thus impossible to make use of the normal zero, an endeavour was made to hit on an arbitrary point or line which would serve as a practicable zero to the

[1] The same appears to be the case with other precipitants of albumen—heat, alcohol, gallic acid, carbolic acid, and bichloride of mercury—all of which I have tried.

scale. After many trials it was found most advantageous to draw the line at a reaction coming into sight midway between half and three quarters of a minute after the addition of the acid : that is, to dilute the urine until it gave no reaction for thirty seconds after the contact of the acid, but showed a distinct opalescence at the forty-fifth second. The exact point aimed at was a reaction coming doubtfully into view at the thirty-fifth to the fortieth second, and appearing still dim, but unmistakable at the forty-fifth second. After a little practice it was found possible to strike this point with sufficient exactness to serve as a practicable zero to the scale.

Each dilution with a volume of water equivalent to the unit volume of urine employed was counted as one degree on the scale; and these degrees might be conveniently termed " degrees of albumen." Thus, a urine which required forty volumes of water to reach the zero reaction might be described as possessing 40 degrees of albumen, a urine requiring three hundred dilutions as possessing 300 degrees of albumen, and so forth.

The difficulty of the method is to hit correctly the zero reaction. When this point is approached, a little more or less dilution makes but a slight difference in the time when the reaction comes into view ; and to obtain exact results—results which compare favorably with those obtained by the weighing process—the testing must be performed in a scrupulously uniform manner. The test-tube must be of a certain fixed diameter, the acid must be added in the right way and at the right moment. The operation should be performed by daylight, or if by gaslight an addition of about 5 per cent. must be made to the result. It is, therefore, necessary to describe the precise mode of operating by which the results which follow were obtained.

The test-tube employed had an interior diameter of five eighths of an inch (15 millimètres). The watch was set on the table before the operator, and beside it was placed the unstoppered bottle of nitric acid. The acid

was added by means of a pipette capable of holding, when immersed to the depth of about two inches, twelve to fifteen drops of acid. When these preliminaries were arranged 5 cc. of the urine to be tested were introduced into a measure graduated to 500 cc., and water was added to 200 or 300 cc.[1] The test-tube was then filled to the depth of about an inch with the diluted urine. The eye was now directed to the watch, and as the seconds hand approached one of the quarter minute strokes (i. e. 15th, 30th, 45th, or 60th) the pipette, previously immersed in the acid and covered with the forefinger, was lifted out and passed into the test-tube, which was held inclined at a wide angle, and on the stroke of the quarter minute its point was pressed against the lower side of the tube, half an inch above the level of the fluid, and the charge of acid delivered. If the manœuvre was properly performed the acid sank to the bottom of the tube and formed a distinct layer below the diluted urine. The test-tube was then held up between the eye and the light against some dark back-ground (such as a dark corner of the room, a book bound in black cloth, or a black sleeve), and as soon as the faintest opalescence appeared above the level of the acid the time of its appearance was noted. If this occurred within thirty seconds after the contact of the acid more water was added and the testing repeated as before. Thus, by repeated additions of water and repeated testing a close approximation to the zero reaction was obtained. A fresh dilution was then prepared, and, guided by the previous trials, two or three more testings with different dilutions were generally sufficient to indicate with exactness the degree of dilution required to produce an opalescence between thirty-five and forty-five seconds after the addition of the acid. If too much water was added in the first instance, and the reaction did not show itself until after the forty-fifth

[1] In ordinary clinical work the fluid drachm is the most convenient unit-volume, and the dilution can be carried out in the imperial pint measure graduated to ounces.

second, the operation was recommenced with less water, and then proceeded with as in the former case.

When the zero-reaction was determined, the degree of dilution required to produce it was noted and expressed in multiples of the unit volume of urine employed. Thus, if 5 c.c. of urine gave the zero reaction when diluted up to 400 c.c., *i.e.* at the eightieth dilution ($\frac{400}{5} = 80$) the urine was registered as possessing 80 degrees of albumen.

When the urine possessed (as often happened) more than 100 degrees of albumen it was subjected to a preliminary dilution with water in the proportion of 1 in 2 or 1 in 5, and the result afterwards multiplied by 2 or 5 as the case might be.

In operating on an untried urine it was first tested in the ordinary way with nitric acid in order to get a rough idea of the degree of dilution likely to be required to reach the zero reaction. The amount and density of the precipitate produced by the acid furnished a useful guide, and saved much time in the subsequent steps of the operation.

The next step in the inquiry was to determine the actual value in weight of albumen of each degree on the scale. This was accomplished by first ascertaining the degrees of albumen by the dilution method and then estimating the quantity of albumen by the weighing process. Selected urines were chosen suitable for the weighing process, and the analyses were performed with all the precautions recommended by Neubauer. These analyses indicated that each degree on the dilution scale corresponded to 0·0034 per cent. of albumen, *i. e.* to 0·0034 grammes of dry albumen in 100 c.c. of urine. The proportion of albumen in a urine was therefore obtained by multiplying the degrees of albumen by the coefficient 0·0034. For example, a urine which possessed 250 degrees of albumen contained 0·85 per cent. of albumen—$250 \times 0\cdot0034 = 0\cdot85$. From these data it was easy to calculate the daily loss of albumen by the urine. Suppose 1200 c.c. of urine to be voided in the twenty-

four hours, and that a sample of this urine showed 250 degrees of albumen, *i. e.* 0·85 per cent., then—

$$\frac{1200}{100} \times 0.85 = 10.2.$$

The daily loss of albumen was 10·2 grammes.

In our English measures the calculation is made as follows :—Suppose, as before, that the urine showed 250 degrees or 0·85 per cent. of albumen, and that the quantity voided in twenty-four hours was forty fluid ounces. Each fluid ounce of the British Pharmacopoeia contains 437·5 grains, then—

$$\frac{437.5}{100} \times 0.85 \times 40 = 148.75.$$

The daily loss of albumen was 148·75 grains.

The time required for the completion of an analysis by the dilution method varied from ten to twenty minutes.

The degree of accuracy possessed by this method may be judged of by the following table, in which the results obtained by it are compared with those obtained by weighing.

Degrees of albumen indicated by the dilution method.		Albumen per cent.		
		Calculated with the coefficient 0·0034.		Found by weighing.
Urine A— 30	...	0·102	...	0·106
B— 98	...	0·333	...	0·308
C—300	...	1·020	...	1·027
D—100	...	0·340	...	0·346
E— 43	...	0·146	...	0·135
F—150	...	0·510	...	0·503
G—116	...	0·394	...	0·381
H—270	...	0·918	...	0·924

The table shows that the dilution method compares favorably with the weighing process even in urines selected for their suitability to the latter process, but it excels it in the diminished time and trouble required for its performance and also in its more general applicability to all grades of albuminous urines. When the proportion of albumen is small the weighing process gives very untrustworthy results, but nearly all albuminous urines

met with clinically are susceptible of a few dilutions before the zero reaction is reached, and are therefore amenable to the method here described.[1]

[1] In the course of the inquiry the question arose whether the relative proportion of the normal urinary ingredients to the quantity of albumen influenced the results. This point was tested by mixing albuminous urines with various known quantities of healthy urine, and then diluting the mixture with water until the zero reaction was reached. These experiments showed that if such mixtures required ten or more dilutions with water to reach the zero the proportion of albumen was exactly indicated, but below this point there was a slow progressive increase of deficiency, or apparent deficiency, in the quantity of albumen indicated. In other words, when the relative proportion of the normal urinary ingredients to the quantity of albumen became very great, the sensitiveness of the nitric-acid test was diminished, and the results came out somewhat below the true values. The error involved is, however, too small to need special correction.

THE UREA AND CHLORIDES IN THE URINE OF JAUNDICE.

BY

J. WICKHAM LEGG, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS AND DEMONSTRATOR OF
MORBID ANATOMY IN ST. BARTHOLOMEW'S HOSPITAL.

I VENTURE to bring before the Society the results of my observations on the urine of patients suffering from jaundice, because I am not acquainted with any like series of observations on the urea and chlorides. A few solitary observations have indeed been published, but it is hard to draw any definite conclusion by comparing them together. Becquerel found the urea in one case to be 17·923 grm. and the quantity 1419 c.c.; in another the urea was 4·057 grm. and the amount 634 c.c. In a pregnant woman, suffering from jaundice, the urea was 7·933 grm. and the amount 640 c.c. in the twenty-four hours.[1] A. Vogel likewise found great decrease of the urea in the urine of a man jaundiced from cancer of the liver : the urea varied from 6·75 to 9·5 grm., the chlorides from 1·6 to 3 grm., while the amount of urine was from 1350 to 2000 c.c.[2] Köllikerand Müller found both in jaundiced dogs and a young

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[1] Becquerel, ' Séméiotique des Urines,' Paris, 1841, pp. 393, 412.

[2] A. Vogel, ' Zeitschrift f. rat. Med.,' 1854, Bd. iv, neue Folge, p. 891.

woman suffering from simple jaundice that the amount of urea was somewhat but not greatly decreased.[1] J. C. Lehmann found, in three cases of jaundice with clay-coloured stools, that the amount of urea was decreased during obstruction to the gall-ducts, but became greater as soon as the obstruction was removed. In a fourth case the same observer found that the amount of urea was increased.[2] Lehmann explains this diminution of the urea by supposing an imperfect digestion of the albuminous. parts of food, probably because the parapeptones are not thrown down. This does not, however, explain the case in which the urea was increased. Leyden records also one of his cases in which the urea daily excreted amounted to 34·8 grm. and even 50 grm.[3] Dr. George Harley, in a case of complete obstruction to the ducts, found the amount of urea to be 27·28, 23·994, and 15·345 grammes on three different occasions, a few days only passing between each analysis. The patient died a fortnight after the last analysis, the jaundice having lasted eighteen months.[4]

Although great attention has been paid of late years to the urine in jaundice, it is somewhat surprising that so few estimations of the urea should have been made. The amount of urea excreted in jaundice is not without interest in discussing Meissner's theory of the formation of urea. For if the formation of urea be one of the many functions of the liver, it might well be looked for that in jaundice, where so many functions are abolished, the urea-forming function would be abolished too. It has been shown that

[1] Kölliker and Müller, 'Verhandlungen der phys-med. Gesellschaft in Würzburg,' 1856, Bd. vi, p. 484.

[2] J. C. Lehmann, 'Ugeskr. for Läger,' 3 R. VI, No. 24—26, abstracted in 'Virchow's Jahresbericht, f., 1868,' Bd. ii, p. 143. Since this paper was read Dr. A. W. Foot has recorded a case of jaundice complicated with xanthelasma, in which the urea excreted in the twenty-four hours was 439·687 grains; quantity of urine 67 oz. ('Dublin Journal of Medical Science,' 1876, May, p. 600).

[3] Leyden, 'Beiträge zur Pathologie des Icterus,' Berlin, 1866, p. 209. .

[4] George Harley, 'Jaundice, its Pathology and Treatment.' Lond., 1863, pp. 74, 78, and 79.

within a few hours of the ligature of the bile-duct, the glycogen disappears from the liver;[1] and this statement has been confirmed by von Wittich.[2] The bile-acid and bile-pigment-forming function of the liver seems to be greatly impaired; the amount of bile-pigment and bile-acids excreted by the kidneys, the only path by which they leave the body, being in the severest cases of jaundice extremely small; if Schwanda's estimations may be trusted, not more than ·014 grm. of bile-pigment in the twenty-four hours;[3] it is notorious how small is the amount of the bile-acids; the smallness of the amount gave rise to their presence in the urine being long denied, and, according to the most competent chemists, the amount excreted in the urine in twenty-four hours never rises to ·5 grm. The amount excreted by the liver in health is certainly not less than 11 grm.; and the difference between health and jaundice has hitherto been explained by supposing the bile-acids to be oxydised in the blood. As they are already in a high degree of oxydation and are exceedingly stable bodies, it seems more probable to believe that very little or no bile-acid is secreted. Indeed, Golowin and other recent observers could find no bile-acids in the urine of persons who had suffered from long-continued jaundice.[4]

Now, most of the supporters of Meissner's theory hold that the glycogen, the bile-acids, and urea are the products of the decomposition in the liver of the albuminous substances of the blood, the carbon, hydrogen, and oxygen going to form glycogen, the oxydised nitrogen and carbon, the refuse of the process, going to form urea and bile-acids. If, therefore, the making of glycogen cease, it would seem likely that the making of bile-acids and urea would cease

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[1] Wickham Legg, 'St. Bartholomew's Hospital Reports,' 1873, vol. ix, p. 161.

[2] Von Wittich, 'Centralblatt f. d. med. Wiss.,' 1875, p. 291.

[3] Schwanda, 'Wiener med. Wochenschrift,' 1865, p. 989.

[4] Golowin, 'Arch. f. path. Anat.,' 1871, Bd. liii, p. 433; and Pye-Smith, 'Trans. of the Path. Soc. of London,' 1878, vol. xxiv, p. 251. See also a paper by the writer on the "Functions of the Liver in Jaundice," 'British Medical Journal,' 1876, vol. ii, p. 263.

likewise. The bile-acids, it is highly probable from the evidence given, do cease to be made. If then the liver in health secrete urea in abundance it might be looked for that in jaundice the amount of urea secreted would fall very greatly in amount or even altogether be brought to nought. The following cases scarcely bear out this inference. In that of Charles Kingsley, in which the obstruction to the ducts was complete and uncomplicated, the amount of urea passed in the twenty-four hours was very little less than 30 grm., that is, about the natural amount. In other cases, in which the urea was diminished, the general health was much affected, and a decrease in the amount of urea might be looked for as a result of the complications rather than as a result of the jaundice.

The observations on the following cases were all made in the wards of St. Bartholomew's Hospital, by the courtesy of the physicians under whose care the patients were placed. The patients were all men, as women show themselves but little fitted for researches of this kind; but beyond this, no selection was made. The cases were taken as they came into the hospital, and I had opportunities of attending to them. The jaundice was deep in seven out of the ten cases. In the others it was not so intense. In all but two the motions were free from colour.

On comparing these cases together it will be seen that the amount of the urine varied very greatly. In two out of the ten the amount was great, the mean being 2234 and 2790 c.c.[1] In eight the amount was neither excessive nor low, the mean varying from 1120 to 1971 c.c. In one the amount was low, the mean being 570 c.c. As to the urea, this bore some superficial relation to the amount of the urine; the highest ranges of urea corresponding to the highest amount of water, and the lowest ranges

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[1] Leyden (op. cit.) has noticed some cases of jaundice in which the amount of urine was much increased. Animals whose bile ducts are tied pass a greatly increased amount of urine, a result noticed by Feltz and Ritter after the injection of bilirubin into the veins. ('Robin's Journal de l'Anatomie,' 1875, t. xi, p. 158.)

of urea corresponding generally to the lowest amount of water. Whether the amount of urea be thought below the natural standard or not depends of course on what is looked upon as the natural standard of health. One observer thinks that 18 grm. of urea in the twenty-four hours do not fall below this standard; and if this be the case, the urea was in one case only below natural, the mean in that case being 15·975 grm. In two out of the remaining nine the urea was 18 grm. in the twenty-four hours; in one 19 grm.; in three 22 grm.; in one 27 grm.; in one 28 grm.; and in the remaining one 29 grm. But if the standard of health be set somewhat higher, as the majority of observers seem to believe, the mean given by Dr. Parkes being 33 grm.,[1] then it is clear that the amount of urea excreted in these cases was, in all, below the standard. It is noteworthy that in the one case in which the excretion of urea was found to be the highest, viz. Charles Kingsley, the obstruction to the bile-ducts was likewise found after death to be complete; more complete, indeed, than in any of the other cases examined; and in the second highest case, in which, however, no examination after death was made, the jaundice had been deep for several years and the stools had been colourless throughout that time.

The chlorides were estimated in nine out of the ten cases, but not so regularly as the urea. In five out of the nine the amount may be said to be almost natural, the mean varying from 13·801 grm. to 10·408 grm. In the remaining four the mean was 9 grm. in two, and 6 grm. in two. In one case the chlorides on one occasion ranged very high, even higher than the urea, being as 21·12 grm. of chlorides to 18·48 grm. of urea. I am disposed to give little heed to this estimation, knowing how abundant chloride of sodium is and how easy the means of deception. The amount of chlorides may be set down as generally of the natural standard.

The diets used at St. Bartholomew's are as follows :—

[1] Parkes, ' The Composition of the Urine,' London, 1860, p. 8.

Full diet.—Two pints of tea, 14 ounces of bread, ½ a pound of dressed meat, ½ a pound of potatoes, 2 pints of beer, and 1 .ounce of butter. Half diet.—Two pints of tea, 12 ounces of bread, 4 ounces of dressed meat, ½ a pound of potatoes, 1 pint of beer, ¾ of an ounce of butter. Milk diet.—Two pints of tea, 12 ounces of bread, 1½ pint of milk, ¾ of an ounce of butter and gruel.

John Harris, aged 30, was admitted into Mark's Ward, under the care of Dr. Duckworth. He had been deeply jaundiced for three years, and had well-marked xanthelasma of the eyelids. The liver was very large, hard and smooth. The spleen was likewise enlarged. The temperature was never raised. The motions were always free from colour. He was out of bed during the day, and allowed to take moderate exercise in the open air. His weight at the end of the observations was 128 lbs. or 58 kilo. The diet from October 20th was half diet, with 4 ounces of wine and arrowroot. From September 8th he took bile capsules three hours after dinner. During the observations on the urine the jaundice was very deep, the complexion of the patient being almost black.

This patient died some time in 1875 at Leicester, but no examination after death took place. Hydatid tumour of the liver pressing on the gall-ducts I consider to have been the most likely diagnosis.

Date. 1873.	Quantity in c.c.	Urea in grm.	Chlorides in grm.
Nov. 9—10	2440	21·35	—
10—11	2370	20·613	12·324
11—12	2040	29·988	15·912
12—18	2060	32·96	14·42
13—14	2140	31·244	13·696
14—15	2340	34·164	12·656
Mean	2234	28·387	13·801

The urine throughout these observations contained a small amount of albumen, that is, it became opalescent

when nitric acid was added to the boiling urine. It also gave a marked Gmelin's reaction. The urine, acid at first passing, readily became alkaline. The estimation of the urea was made by Liebig's process; that of the chlorides by the volumetric method with nitrate of silver after incineration with nitrate of potash.

Charles Kingsley, aged 35, was admitted on November 18th, 1873, into Matthew's Ward, under the care of Dr. Black. He became jaundiced a twelvemonth before admission. He was deeply jaundiced, and the stools were white. The liver could be felt, smooth and firm, reaching down to the umbilicus. During the observations on the urine he was allowed to walk about the ward. The temperature varied from 97° to 99° F.

He died on December 5th, the temperature for forty-eight hours before death being as high as 105° or 106°. A large hydatid cyst pressed on the common duct, completely obstructing it.[1]

Date. 1873.	Quantity in c.c.	Urea per cent.	Chlorides in grm.
Nov. 20—21	2840	1·04	11·36
25—26	1430	·88	—
A good deal lost.			
26—27	2740	1·08	—
Mean of 2 obs.	2790	1·06	11·36
		or 29·574 daily.	

This patient would not collect all his urine. It was throughout opalescent with heat and nitric acid, showing a distinct Gmelin's reaction with nitric acid. The specific gravity varied from 1008 to 1012. The estimation of the urea was by Liebig's process; that of the chlorides after incineration with nitrate of potash.

James Kean, aged 50, was admitted on November 21st, 1873, into Luke's Ward, under the care of Dr. Southey. About nineteen months ago he was first seized with the

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[1] This case has been given in full in the 'Transactions of the Pathological Society of London,' vol. xxv, 1874, p. 155.

jaundice, attended with attacks of sharp pain in the right hypochondrium, coming on about six times a day. The jaundice lasted a twelvemonth. He has now been jaundiced since November 18th. At the very beginning of the observations he was recovering from an attack of pneumonia, but the temperature throughout this attack and during the observations was never higher than 98·4° F. The man was in bed during the observations; the liver large, stretching down to within an inch of the umbilicus; the stools half fluid, all brown. The diet from November 21st was milk diet, with arrowroot and half a pint of beef tea. The medicine was a draught of effervescing tartrate of soda, with ten minims of spirits of chloroform every six hours.

He continued under observation till February, 1874, being still somewhat jaundiced, but with the liver greatly decreased in size.

Date. 1873.	Quantity in c.c.	Sp. gr.	Urea in grm.	Chlorides in grm.
Nov. 25—26	... 2430	... 1010	... 21·384	... —
26—27	... Lost			
27—28	... 1970	... 1013	... 24·822	... —
28—29	... 1570	... 1015	... 24·492	... —
29—30	... 1445	... 1022	... 32·079	... —
30—Dec.1	... 1865	... 1015	... 28·348	... 13·001
Dec. 1—2	... 1435	... 1020·5	... 32·321	... 13·489
2—3	... 1780	... 1015	... 29·836	... 10·918
3—4	... 1590	... 1019	... 34·98	... —
Mean	... 1788	... —	... 27·28	... 12·469

On the first three days of observation the urine was alkaline at the time of testing; on the first two days and on the last it became opalescent upon the action of heat and nitric acid; on the other days, not. Gmelin's reaction was present throughout. The urea was estimated by Liebig's method; the chlorides after incineration with nitrate of potash.

Thomas David Smart, aged 42, was admitted into John's Ward, under the care of Dr. Harris, on November

24th, 1873. He had been jaundiced for about three weeks; vomiting preceding the jaundice. Jaundice on November 28th was universal, but not very intense; stools white. Liver not to be felt; liver dulness about four inches vertically in nipple line; nothing else noteworthy in the belly. The motions became of a pale brown colour on November 30th; on December 3rd of a natural brown. The yellowness was much less on December 4th, and he was discharged well on December 20th. The temperature taken daily in the mouth during the observations on the urine never rose above 100° F., nor sank below 99° F.

Date. 1873.	Quantity in c.c.	Sp. gr.	Urea in grm.	Chlorides in grm.
Nov. 28—29	1665	1014	26·64	—
29—30	1400	1018	22·96	—
30—Dec.1	1300	1020	21·45	—
A good half-pint was lost.				
Dec. 1—2	1225	1019	22·54	9·555
2—3	1310	1018·5	25·021	9·17
3—4	1450	1013·5	19·43	9·96
4—5	1085	—	18·9875	13·02
Mean	1355	—	22·596	10·428

The urine contained no sugar throughout the observations; it was opalescent with heat and nitric acid, save on the last two days. Gmelin's reaction was present throughout, becoming fainter on the last two days. The urea was estimated by Liebig's process; the chlorides after incineration with nitrate of potash.

It is noteworthy that in this case the urea decreased on the last two days of observation, as the motions assumed a more natural colour.

William King, aged 46, was admitted on August 3rd, 1874, into Radcliffe Ward, under the care of Dr. Andrew. He was quite well until last January, when he was seized with a griping pain in the belly, which has come on now and then since. Fourteen days before admission he

became jaundiced. During the observations he was deeply jaundiced. His pulse was 64. His temperature did not rise above 98°. His fæces were colourless. His diet on September 17th and throughout the observations on the urine was milk diet and a fish daily, with a pint of arrowroot, two eggs, a pint of mutton broth, and two ounces of brandy. To this a pint of milk was added, September 22nd. He took the nitro-muriatic acid mixture of the Pharmacopœia three times a day, with a bile capsule three times a day two hours after food.

He died on November 6th. The common duct was found pressed upon by a tumour from the pancreas, and was dilated above. The tumour of the pancreas and multiple tumours of the liver were carcinomatous.[1]

Date. 1874.	Quantity in cc.	Sp. gr.	Urea in grm.	Chlorides in grm
Sept. 17—18	620	1025	18·662	7·376
18—19	490	—	17·376	4·165
20—21	540	1028	20·388	5·994
21—22	425	—	15·0025	5·015
22—23	770	1026	23·87	9·317
23—24	580	1025	17·922	5·394
Mean	570	—	18·87	6·21

The urine was acid throughout the observations, and became slightly opalescent with heat and nitric acid. He was deeply jaundiced throughout. The urea was estimated by Liebig's process; the chlorides directly, without incineration.

Robert Bristow, aged 38, was admitted on May 15th, 1875, into John's Ward, under the care of Dr. Church. He had been jaundiced for five years before admission. The liver descended four inches below the ribs in the nipple line, and a round swelling could be felt on it. The spleen filled the left hypochondrium. He was not very deeply jaundiced, and the stools were not free from colour.

[1] A full report of this case has been published in the 'St. Bartholomew's Hospital Reports,' 1875, vol. xi, p. 82.

The patient was allowed to get up, and temperature was natural during the observations on the urine. He was ordered full diet with a pint of milk. On June 14th a bottle of soda water daily was ordered. From May 29th he had twenty minims of compound tincture of cardamoms, fifteen grains of bicarbonate of soda in an ounce of compound infusion of gentian. On the 14th of June this was changed to three grains of quinine thrice daily.

Date. 1875.	Quantity in cc.	Sp. gr.	Urea in grm.
June 16—17	... 1210	... 1019	... 23·595
17—18	... 1680	... 1012	... 23·52
18—19	... 1400	... 1014	... 21·00
19—20	... Lost		
20—21	... 1340	... 1015	... 21·44
21—22	... 1340	... 1015	... 21·44
22—23	... 1640	... 1014	... 24·60
Mean	... 1483	... —	... 22·598

The urine became slightly opalescent on the first two days of observation with heat and nitric acid, not on the remaining. There was throughout a deep Gmelin's reaction with nitric acid. The urea was estimated on Russell and West's method.[1]

Thomas Johnson, aged 49, was admitted on July 19th, 1875, into Mark's Ward, under the care of Dr. Andrew. Twelve weeks before admission he was quite well. Six weeks before admission he became jaundiced. Liver on admission was enlarged, stretching two to two and a half inches below ribs. Motions perfectly free from colour, throughout his stay in hospital. Temperature varied from $98·5°$ to $99·4°$; pulse from 84 to 100, and he was in bed during the observations. On admission he was ordered milk diet and arrowroot. A pint of beef tea was ordered on the 23rd. A warm bath was given on July 23rd. The medicine given was the alkaline calumba draught of

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[1] This method is described at full in the 'Journal of the Chemical Society,' 1874, vol. xxvii, p. 749.

St. Bartholomew's Hospital Pharmacopœia, with ten minims of tincture of opium every six hours, and occasional doses of chloral (gr. xv). Death took place on August 21st, after a rise of temperature for four days before death as high as to 104°. The cause of the jaundice was cancer of the pancreas; nodules were likewise scattered through the liver, with great dilatation of the gall-ducts and bladder, and abundance of gall-stones in the gall-bladder.

Date. 1875.	Quantity in cc.	Sp. gr.	Urea in grm.	Chlorides in grm.
July 22—23	... 1920	... 1010	... 21·12	... 4·976
23—24	... 2090	... 1010	... 22·99	... 7·106
24—28	... Diarrhœa			
28—29	... 2450	... 1010	... 30·40	... 9·02
29—30	... 1840	... 1010	... 21·16	... 6·624
30—31	... 2160	... 1010	... 25·92	... 8·208
Aug. 1—2	... 1370	... 1011	... 15·67	... 4·658
Mean	... 1971	... —	... 22·87	... 6·765

Throughout these observations the urine was acid, clear, of a deep brown-green colour, and free from albumen and sugar, Gmelin's reaction being well marked. The estimation of the urea was made by Russell and West's process; that of the chlorides without incineration.

Robert Langan, aged 47, was admitted on July 29th, 1875, into Mark's ward, under the care of Dr. Andrew. He became jaundiced two months before admission for the first time in his life; he had never any pain in the belly. No enlargement of liver or spleen; no ascites. He says he has always been sober, but is easily overcome by liquor. On first rising he is accustomed to bring up a little yellow phlegm. Stools of light brown colour, particoloured. On August 3rd the stools became ashy grey, and the jaundice was noted to be less. From admission up to August 1st he was on milk diet with beef tea, changed at that date to milk diet with a pint of milk and arrowroot.

A fish daily was added on the 5th. The patient was kept in bed. The temperature varied from 97·2° to 98° F. Pulse from 60 to 72. He had a warm bath on the 3rd and 6th. For medicine he had the draught of nitro-muriatic acid of the St. Bartholomew's Hospital Pharmacopœia, with gr. xx of sulphate of magnesia three times a day. He was discharged from the hospital on September 15th, no material change having taken place. He was again seen on January 24th, 1876, no great change having taken place in the symptoms or physical signs, save a considerable increase of the jaundice during the fortnight before being again seen.

Date. 1875.	Quantity in cc.	Sp. gr.	Urea in grm.	Chlorides in grm.
Aug. 1—2	... 1760	... 1017	... 18·48	... 21·12
2—3	... 1710	... 1013	... 17·1	... 14·364
3—4	... 1240	... 1015	... 12·4	... 7·728
4—5	... 1130	... 1018	... 14·69	... 10·17
5—6	... 1565	... 1015·5	... 17·215	... 12·9895
Mean	... 1481	... —	... 15·975	... 13·2743

The urine was acid throughout these observations, free from sugar, but containing bile pigment, and becoming opalescent with heat and nitric acid. The urea was estimated by Russell and West's method; the chlorides without incineration.

Josias Davidson, aged 48, was admitted into John's Ward, under the care of Dr. Church, on October 23rd, 1875. He began to be jaundiced about last May. On admission he was deeply jaundiced, the stools colourless, the liver large, but surface smooth. His temperature during the observations on the urine was 98°. He had full diet with a pint of milk. He took the nitro-muriatic acid draught of the St. Bartholomew's Hospital Pharmacopœia three times a day, with twenty minims of the Sp. Chloroform. He was in bed during the obser-

vations on the urine. He died on December 12th. Numerous white nodules of cancer were found in the liver; the common duct greatly dilated, but a probe could be passed down it into the gut; no bile in the gut. The head of the pancreas was a soft white tumour, pressing on the common duct.

Date. 1875.	Quantity in cc.	Urea in grm.	Chlorides in grm.
Oct. 24—25	... 1150	... 20·7	... 8·05
25—26	... 1100	... 19·8	... 8·14
26—27	... 1120	... 20·16	... 10·08
27—28	... 1300	... 22·1	... 13·26
28—29	... 1190	... 17·85	... 11·186
29—30	... 850	... 12·70	... 7·82
	Some lost	or 1·4 p. c.	
Mean	... 1120	... 18·72	... 9·756

The urine was acid throughout, opalescent with heat and nitric acid, turbid on the first and last days of observation, on other days clear; it was high coloured throughout, giving Gmelin's reaction readily. The urea was estimated by Russell and West's process. The chlorides without incineration.

Frederick Sheffield, aged 53, admitted into Mark's Ward, under the care of Dr. Andrew, on November 26th, 1875. He had been feeling out of sorts for several months before admission. The jaundice had been noticed for three weeks before admission. On December 1st he was ordered full diet with arrowroot. The motions remained free from colour during observation, but the jaundice was never very deep. The temperature was never above 98·2°, and his weight was 128 lbs. or 57 kilogrammes.

He was discharged from the hospital on January 1st, 1876.

Date. 1875.	Quantity in cc.	Sp. gr.	Urea in grm.	Chlorides in grm.
Dec. 14—15	.. 930 ...	1021	... 13·02 ...	6·51
	Some lost		or 1·4 p. c.	or ·7 p. c.
15—16	... 420 ...	1018	... 9·24 ...	3·528
	Some lost		or 2 2 p. c.	or ·84 p. c.
16—17	... 1190 ...	1019	... 15·47 ...	9·996
17—18	... 1100 ...	1017	... 19·8 ...	9·78
18—19	... 1070 ...	1023	... 19·26 ...	9·436
19—20	... 980 ...	1015	... 12·74 ...	6·272
	Some lost		or 1·3 p. c.	or ·64 p. c.

The urine was acid throughout, and showed abundant evidence of bile pigment. The urea was estimated by Russell and West's process ; the chlorides after incineration with nitrate of potash.

ON

SOME EFFECTS OF LUNG ELASTICITY IN HEALTH AND DISEASE.

BY

R. DOUGLAS POWELL, M.D., F.R.C.P.,

PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION AND DISEASES OF THE
CHEST AT BROMPTON; SENIOR ASSISTANT PHYSICIAN TO THE
CHARING CROSS HOSPITAL.

THERE is, perhaps, no department of clinical medicine which has from time to time been worked at with greater success, yet in which greater errors of a physiological kind still prevail, than the department of chest diseases. The classical paper of John Hutchinson communicated to this Society in 1846 remains unsurpassed in the interest and importance of the facts related; his spirometer may still be found lurking in some consulting rooms, and its teachings will ever remain a part of our knowledge, although the instrument itself is replaced by other more convenient and discriminating methods of physical diagnosis.

Mr. Hutchinson's doctrine respecting the mechanism of respiration may still be said to be the current doctrine at the present time. He taught that ordinary inspiration was effected mainly by the muscular action of the diaphragm, that deep inspiration was effected, on the other hand, mainly by the expansion of the chest by mus-

cular action, and that in each case the forces to be contended against were the elasticity of the lung and the elasticity of the chest-walls. Twenty years later, however, in a most able series of lectures delivered at the College of Physicians, Dr. Hyde Salter made one important modification of Hutchinson's teaching. In these lectures upon the causes of dyspnœa,[1] Dr. Salter contended that the mechanical process of respiration might be considered as starting from a position of equilibrium at the end of ordinary inspiration, in which the thoracic parietes and diaphragm had followed the recoil of the lung to that point at which lung tension was equalled by thoracic resilience. At the commencement of inspiration, therefore, the thoracic elasticity was, Dr. Salter maintained, a force acting in favour of, and not against, expansion. As inspiration proceeded, however, thoracic resilience became an expiratory force acting *with* lung tension against further expansion, and, finally, these two elastic forces served, on muscular action ceasing, to effect expiration. I have copied the tabular form in which Salter placed his views with some slight alteration of arrangement.

Coefficients of Respiratory Statics.

At level of—	Forces tending to expiration.	Equilibrium	Forces tending to inspiration.
A. Ordinary expiration	{ Elasticity of lung / Gravity of chest-wall		Elasticity of costal parietes (*excentric thoracic resilience*).
B. Ordinary inspiration	{ Elasticity of lung / Elasticity of parietes / Gravity of chest-wall		Muscles of ordinary inspiration, diaphragm principally.
C. Extreme inspiration	{ Elasticity of lung / Physical reaction of parietes (*concentric thoracic resilience*) / Gravitation of chest-wall	Extreme	Muscles of extraordinary inspiration.
D. Extreme expiration	{ Elasticity of lung / Gravity of chest-wall / Muscles of extraordinary expiration		Elasticity of costal parietes, excentric thoracic resilience (extreme).

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[1] Vide 'Lancet,' Aug. 5th, 1865.

The best modern physiologists now recognise the fact that in the normal position of thoracic repose the contractility of the lungs is exactly counterpoised by the elastic resilience of the chest-wall. [We may accurately name this elastic resilience of the chest-wall the *excentric thoracic resilience*.] Dr. Burdon Sanderson, in the 'Handbook for the Physiological Laboratory,' p. 294, speaks thus of excentric thoracic resilience; he observes that " The muscular movements by which the chest is expanded must be studied in their relation to a certain definable position of the thorax which is called *the position of equilibrium*. It is the position assumed by it at the end of normal expiration. For as no muscle takes part in the normal expiratory act, the whole thoracic muscular apparatus is at that moment in a state of rest, the bones and cartilages assuming that position which results from the balance of the opposed elastic forces which act upon them from within and from without. Of these elastic forces the most important is that of the lungs, which organs, being contained in a cavity much larger than they are themselves, to the inner surface of which their external surface is inseparably applied, constantly draw together its walls with a force to be investigated in a future paragraph.

" Next in importance is the elasticity of the ribs and cartilages, by virtue of which the thoracic wall ever tends to be larger than it is, in opposition to the contractile influence of the lungs." That the thoracic parietes are drawn inwards by the elastic recoil of the lungs would scarcely seem to need demonstration; it is demonstrable, however, by an experiment of Dr. Salter's.[1] He took a capillary tube expanded at its lower end into a bell shape and closed at this extremity by a thin layer of caoutchouc. Filling the bell-end with a coloured fluid so as to include a few inches of the tube, he fixed the instrument vertically, so that the caoutchouc touched the cartilage of the third rib of a man recently dead. The integuments had pre-

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[1] Loc. cit., p. 143.

viously been reflected from the chest. He now cautiously divided the cartilage, but no movement of the fluid took place. On penetrating further, however, into the pleural cavity air was heard to enter the chest, and the fluid mounted the tube to the height of one additional inch. This movement Dr. Salter estimated to be equivalent to an expansion of $\frac{1}{100}$ of an inch.

In May, 1871, Mr. Le Gros Clark related, in a paper read before the Royal Society, an experiment to demonstrate what he terms "the passive tension of the diaphragm." "The trachea of a sheep immediately after death was exposed in the neck, divided, and tied over a glass tube, which was put in communication with a graduated receiver placed under water and guarded by a stopcock. The pleuræ were then opened, and, as air entered, the diaphragm became flaccid, but the lungs remained unchanged in position and form." It is clear that the air in this experiment could only enter the pleura in consequence of the expansion of the chest by virtue of the *excentric thoracic resilience*. For, the lungs being secured, they could not collapse and so suck in the air. Mr. Le Gros Clark's experiment is further valuable to the present purpose inasmuch as it very clearly shows that the diaphragm also exercises some elastic traction upon the lung; indeed, it is Mr. Clark's object in his paper to point out that this *passive tension* is the means of retaining the supplemental air in the lungs.

My principal object in bringing the present paper before the Society is to endeavour to point out some of the bearings of these physiological facts in clinical medicine, and to indicate how they serve to afford us a better insight as to the true mechanism and relative value in diagnosis of some of the signs of chest disease. Let me, however, venture first to observe that, so far as I am aware, no physiologist has fully pointed out the importance of the thoracic resilience as a conservator of force in respiration. Mr. Hutchinson and all who have followed him place thoracic elasticity as a force, the chief force indeed, against

which the inspiratory muscles have to contend, and which is wholly in favour of expiration. Even Dr. Salter, who observes that thoracic elasticity is in favour of ordinary inspiration at its very commencement, places it as the opponent of the latter portion of that act. It is obvious, however, on very slight reflection, that the elasticity of the chest-wall is a force, not only in favour of inspiration at the commencement, but against expiration at the termination of the respiratory act. It renders easier the expansion of the chest by neutralising the first resistance and inertia of the lungs, and in the final contraction of the chest in expiration exercises a buffer-like action in taking off the shock of recoil. This elastic *help* at the beginning and elastic *kindrance* at the end of the respiratory act is a spring-like function of the chest-wall, the importance of which has not been duly acknowledged. It is only necessary, however, to glance at the emphysematous chest to note the uneasiness entailed by its loss. The lungs in emphysema have not lost all their elasticity, but they have lost so much of it that the excentric thoracic and diaphragmatic resilience has nothing to oppose it, the chest boundaries are no longer therefore drawn inwards by the lungs in expiration beyond the position they would of their own accord adopt. Every physician must have repeatedly remarked the peculiar jerk or tug with which inspiration commences in extreme emphysema, a *tug* which communicates a shock to the stethoscope very appreciable to the ear.

It is important to know whether in calm breathing the chest-wall expands beyond the point to which its resilience would, if unopposed by the lung-elasticity, carry it. Mr. Hutchinson calculated the costal movement in health not to exceed from $\frac{1}{16}$ to $\frac{1}{4}$ of a line, *i. e.* from 1—2 millimètres in quiet breathing. Taking the average of Dr. Sanderson's measurements in the case of a healthy man aged 22, we find the expansion to be 1.6 millimètre.

Dr. Sibson gives the thoracic movement during tranquil

inspiration as $\frac{1}{20}$ inch; in very robust people rather less, in weakly persons considerably more.

Dr. Ransome, in his valuable paper on the respiratory movements read before the Society in 1872, has, unfortunately, not recorded the thoracic movement of calm breathing.

I have myself made some observations to ascertain the extent of thoracic expansion in calm breathing by means of a very simple but accurate instrument made for me by Mr. Hawksley. This instrument may be briefly described as consisting of a convenient chest-piece connected with a straight rod made of a light material by means of an elastic joint. The other end of the rod has a metallic writer affixed which marks upon a horizontal plate that can be shifted by ordinary clockwork. This instrument simply records the forward movement of the chest-wall in any given direction, without reference to the mechanism in such expansion which does not, for the moment, concern us.

The mean of nineteen measurements of expansion taken from myself, instrument being applied at mid-sternum level of fifth cartilage was 2·75 mm., least expansion being 2·5, greatest 4 mm. Observations upon others have shown considerable variations, but within this range.

Let us now compare these movements with the expansion of the chest as ascertained *post mortem* to be the result of its resilience alone. Dr. Salter, in his experiment upon the dead subject already quoted, measured an expansion of the chest on permitting air to enter the pleura of 1 millimètre. I have repeated Dr. Salter's experiment at different points of the chest, avoiding, however, in all cases dividing any of the cartilages.

The instrument used in these experiments is similar to that employed by Dr. Salter, and consists of a fine tube (A, B) expanded at its lower end into a bell shape, across which is stretched a piece of caoutchouc (B) having attached to its centre a projecting button (C). The bell is filled with a coloured fluid, which occupies also a few inches of

Fig. 1.

the fine tube. Diameter of base of tube B is found to be 22 millimètres. Diameter of tube 1·5 mm., their proportion to one another being therefore as 14·66 to 1.

Experiment 1 (*vide* Table No. I).—Male subject, aged 33, death one week after double amputation for gangrene.

Post-mortem, twenty-four hours. — Rigor mortis still present in arms, slight tinge of commencing decomposition at root of neck. Right side of chest perceptibly more prominent than left.

Integuments reflected from chest, trachea cut across below cricoid cartilage, to ensure no obstruction from any mucous collection at the glottis. The third cartilage exposed on each side of the sternum. Instrument fixed by suitable holder vertically over the third left cartilage, so that button of bulb rested lightly upon it, causing the level of fluid in the tube to rise a few inches. The level of the

fluid D in the stem then accurately noted from scale attached, and found to be quite sensitive to any expansion or recession of the chest-wall. The pleura was then cautiously opened by an incision between the second and third left cartilages, and the fluid in the stem (observed by Dr. Coupland) steadily but somewhat slowly rose 1¼ inch, or 3·15 centimètres, which, divided by 14·6, gives 2·143 millemètres of actual expansion. On opening the right pleura in similar way, no further expansion. On examining the chest, the cartilages were not ossified, both lungs were somewhat emphysematous, right more so than left.

My results are briefly recorded in the following table (see Table No 1).

Insufficient as these observations are to justify any very positive conclusions, they yet furnish strong evidence in favour of the two following propositions :

1. That in health the excentric thoracic resilience favours inspiration throughout the normal respiratory act in calm breathing.

2. That in emphysema or œdema, as the lungs enlarge, the thoracic walls yield outwards, but that up to an expansion of probably between 3 and 4 millimètres there is no intrathoracic pressure.

I must not omit here to thank Dr. Coupland for his kind help in giving me opportunities for my experiments at the Middlesex Hospital, and for co-operating with me in making the observations.

Ordinary inspiration being mainly, however, effected by the descent of the diaphragm, the resilience of that muscular membrane, its "passive tension" according to Mr. Le Gros Clark, is in favour of inspiration at the commencement of that act. This resilience of the diaphragm is doubtless derived from its attachment to the costal margin. It is usually said that the diaphragm is the only muscle used in calm inspiration; it is obvious, however, that if this were so the chest-walls would yield inwards with the descent of this muscle; whereas, on the contrary, the chest expands a little, albeit a very little, and

TABLE I.—*Giving observations on thoracic resilience in 10 cases post mortem.*

All observations made with the co-operation of Dr. Coupland at the Middlesex Hospital.

this must be by the aid of muscular action. The inter-costals especially, as pointed out by Mr. Clark, acting from above downwards, *i. e.* from the less to the more movable ribs, support and even expand the costal margin whilst it endures the muscular traction of the diaphragm. It is the function of the excentric thoracic resilience to reduce the muscular power thus used in calm inspiration to a minimum.

A word or two must now be said as to the manner in which the mediastinum is affected by the elastic tension of the lungs. Dividing the thoracic cavity into two halves, the mediastinum presents a surface to each, and between these two surfaces are included the heart and the great vessels proceeding to and from it, the main bronchi, glands, &c. The mediastinum is movable from side to side within certain limits. Now, just as the elastic tension of the lung draws upon the thoracic wall, and has as its counterpoise the resilience of the ribs and cartilage, so, in the median line, the elastic lung on one side draws upon the mediastinum and has as its counterpoise an equal traction of the opposite lung. This traction upon the mediastinum from either side is *constant* in health, existing even in extreme expiration, but it is vastly increased with the expansion of the lungs. Its tendency is to draw apart the two layers, if we may so think of them, of the me-diastinum and to exercise a suction power upon the blood towards the heart, arteries, and veins which they include. This constant determination of blood towards the heart, increased with each inspiration, has attracted much atten-tion. It is beyond our purpose now to consider its mul-tiple effects and counter-effects. It is obvious, however, and the stethograph records the fact, that every contrac-tion of the heart modifies the respiratory curve, and, con-trariwise, the respiration modifies the pulse-trace. We need go no further to find the chief cause of the constant tendency to the entry of air into a vein opened at the root of the neck.

The above considerations enable us better to understand

some phenomena of disease which would be otherwise obscure, and which have been in some instances inaccurately or inadequately explained.

Among the diseases which affect the lung- or thoracic elasticity we must first speak of that common pulmonary disease *vesicular emphysema* of the so-called hypertrophous kind. It has been currently taught since the time of Stokes that the characteristic signs of emphysema—the enlarged chest, the displaced heart, the lowered diaphragm and abdominal organs, and the troubled circulation—are due to the pressure of the expanded lungs. Now, it would be unwise to assert that such pressure by the lungs upon their surroundings is *impossible*, but both physiological and clinical observation, I think, concur in showing that it almost never occurs. It is singular that Stokes[1]—who regarded the lungs in emphysema as being compressed by the chest-walls, and as in their turn pressing upon other parts—should have at the same time given it as his constant experience that the intercostal spaces, so far from being bulged, are, in fact, more deeply marked in this disease than in health.[2] Without going so far as Dr. Stokes, whose assertion in this latter respect has been earnestly combated by others, one may safely affirm that only in very marked cases of emphysema do the intercostal spaces become *level* in expiration, that save in forced expiration these spaces never puff outwards, and that they invariably deepen at the first moment of inspiration. Similarly do all other soft parts deepen with inspiration even in the most extreme cases of emphysema. This is very notably the case with the supra-clavicular and supra-sternal fossæ. These facts prove that although the lungs in emphysema are expanded from loss of their elasticity, according to the degree of emphysema, the chest is similarly and to an equal extent expanded, not by any *pressure of the lungs*, but by the *excentric thoracic resilience* being less and less

[1] 'Dis. of Chest,' 1837, part i, p. 187.

[2] Dr. Stokes observes that, even in fluid effusions, the intercostal grooves are not obliterated, except in very severe cases or in cases of long duration.

opposed as the emphysema increases. Any further en-
largement of the chest during inspiration, however, is
opposed, as it is in health, by the elasticity of the lungs
and, I may add, by the *con*centric thoracic resilience. In
fact, as has been pointed out by Dr. C. J. B. Williams and
others, inspiration in great emphysema begins at the level
at which normal inspiration would terminate, *i. e.* between
the levels B and C of the table of coefficients. The dia-
phragm is in emphysema similarly lowered, not being
drawn up to its normal degree of convexity with expira-
tion; it is hence disabled for inspiration, which is mainly
accomplished by thoracic expansion in this disease in its
more advanced stages. The extraordinary muscles of in-
spiration are habitually used in emphysema, and the
peculiar jerk with which they come into action, at once to
overcome the inertia and resistance of the thoracic parietes
and the lungs, has already been referred to as illustrating,
by contrast, the important part elasticity plays in facilitat-
ing the normal respiratory act. The position of the heart
and liver are necessarily lowered with the lowered position
of the diaphragm. It is inaccurate, therefore, to say that
these organs are pushed downwards by the pressure of the
enlarged lungs. It must, too, be only in very rare in-
stances that the heart is compressed between the lungs.
These organs are sometimes, however, described as, in
emphysema, bulging forth after death on removing the
sternum. My own observation would lead me to doubt
this occurrence except where some fluid is present in the
pleura, or some obstruction such as inhaled blood-clot
occludes bronchi immediately before death. The whole
venous circulation is in emphysema impeded by the ex-
tinction of that *constant* aspiration towards the heart which
obtains in health, and which in this disease is only present
in an enfeebled degree during inspiration. Sir William
Jenner has, however, clearly pointed out that the obstruc-
tion in the pulmonary capillaries is the chief cause of the
disordered circulation and dropsy that ensue in this disease.

In another condition of things the opposite to that of

emphysema, viz. in which, while the lung elasticity remains normal, the thoracic wall has suffered loss of resilience through impairment in the nutrition of its bony and muscular structures, we observe recession of the least resisting parts, increased during inspiration, and consequent deformity of the chest.

It would appear from some interesting observations by M. Woillez[1] that in the pyrexial stage of acute diseases an appreciable enlargement of the thoracic circumference takes place, gradually to subside as the fever abates. M. Woillez has taken careful measurements in cases of variola, idiopathic erysipelas, scarlatina, articular rheumatism, pneumonia, hæmoptysis, bronchitis, and pericarditis, and he finds the enlargement to take place within the first few hours of the onset of fever, and to more gradually subside to the normal as the disease retrogresses. He considers the increased circumference of the chest to be due to a " diminished general elasticity of the chest," which he traces mainly to loss of elasticity of the lungs in consequence of their temporary engorgement in fever. I do not know that any one has repeated these observations of M. Woillez.

The ready expansion of the chest-walls by virtue of their elastic resilience to the increased capacity needed in all cases of acute and rapid enlargement of one or both lungs, e. g. in pneumonia, bronchitis, whooping-cough, &c., is a beautiful instance of adaptation in the human mechanism.

In cases of effusion into the pleura, whether of air or fluid, there are two phenomena to be always observed, unless their occurrence be prevented in some exceptional manner, viz. displacement of heart and enlargement of the circumference of the chest, especially of that half of it corresponding with the affected side.

Displacement of the heart in pneumothorax. — On the occurrence of pneumothorax the condition of the air within the pleural cavity as regards pressure depends upon the nature of the perforation, whether it be a free or a

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[1] " Recherches sur les Variations de la Capacité thoracique dans les Maladies Aigues," 'Mémoires de la Société Médicale d'Observation.'

valvular opening. If the communication with the bronchus be free and patent, the air passes to and fro through the lung with inspiration and expiration. In this case it is obvious, and it can be readily shown, that there is no compression of the air within the pleura. If, however, the perforation be not free, but more or less valvular, the entry of additional air into the pleura readily takes place, but its escape with expiration is imperfect or impossible, and consequently there is in these cases air pent up in the pleura and exercising pressure upon all surrounding parts.

Now, as a matter of clinical experience, we find in both cases, *i. e.* when there is free circulation of air in the pleura and when the air is confined and pent up there, that displacement of the heart is one of the first and most necessary signs of the pneumothorax.

I have tabulated some notes of seventeen cases of pneumothorax to give information with regard to the degree of pressure and the position of the heart. Of these 17 cases 9 were males, 6 females, and in 2 instances the sex is not stated. The pneumothorax was in 5 cases on the right side, in 10 on the left, and in 1 case it was double-sided. In 12 of the cases tabulated there was found *post mortem* an air pressure within the pleura varying in degree :—the highest pressures being 7 inches, $5\frac{1}{2}$ inches, and $5\frac{3}{10}$ inches of *water* respectively, and the lowest pressures $1\frac{3}{4}$ inch and 2 inches. In all these cases the heart was displaced, although in four of them I do not find the fact definitely stated in my notes. Woodcuts Nos. 2 and 3 are diagrams of a case, No. 11, in which the intra-pleural pressure was the highest. One diagram (woodcut No. 2) was taken during life and illustrates the clinical features of the case ; the other (No. 3) delineates the position of parts found at the *post-mortem* examination.

In 4 out of the 17 cases there was no intra-pleural pressure, yet in 3 of these cases the heart was very greatly displaced, and in the fourth case, in which the heart was not displaced, this was accounted for by the unruptured lung being so consolidated as not to permit of its collapse.

TABLE II.—*Cases of pneumothorax tabulated.*

P.-M. hours.	Air pressure in pleura.		Physician.	Remarks.
30	4 in. of water		....	No further p.m. notes.
12	5¼ in. „	Displaced to left	Dr. Sanderson	Two openings in pleura ; no decomposition or distension of abdomen ; ¾ pint of turbid fluid in pleura.
14	2 in. „	Displaced to right	Dr. Powell	No examination allowed beyond testing pressure.
16	3½ in. „	Not stated	Dr. Sanderson	Opening valvular.
30	2 in. „	Ditto	Ditto	Base of ... d—the only healthy porti
30	4 in. „	Displaced to left	Dr. Quain	Liver displaced downwards ; opening perfectly valvular. *Vide* ' Path. Trans.,' vol. xix, p. 77.
20	1¼ in. „	Displaced backwards	Ditto	Air from left pleura had entered into pericardinm. ' Path. Trans.,' vol. xx, p. 99.
24	L. 3½ in. „ R. 2½ in. „	On admission displaced to right	Dr. Pollock	Two quarts of pus in left pleura.
30	Nil	Displaced to right	Dr. Cotton	Left pleura contained a small quantity of purulent fluid. ' Med. Times,' Aug. 21st, 1869.
30	Nil	Displaced to right of sternum Ditto	Dr. Alison	Left pleura contained about a pint of purulent fluid. Loc. cit.
18	7 in. „	Ditto	Dr. Pollock	A valvular opening.
...	2 in. „	Not stated	...	Opening valvular.
30	3¾ in. „	Displaced to right	Dr. Pollock	Opening could not be discovered.
...	Nil	Not displaced	Dr. Cotton	Recent c
...	Not noted	Ditto	Dr. Pollock	Opening lung c
40	5½ in. „	Displaced to left	....	Small valvular opening.
...	Nil	Apex at left ensiform	Dr. Cotton	Opening free. *nil ; right*

SUMMARY.—17 cases { 9 M. 6 F. 2 ? } { 6 Right 10 Left 1 Double } { 12 pressure from 1¼ to 7 in. 4 no pressure 1 not noted } { 8 heart displaced 4 not stated } Probably in all cases displaced. { 3 heart displaced. 1 not displaced, accounted for by consolidation of left lung. Heart not displaced, right lung consolidated.

Fig. 2.   Fig. 3.

Figs. 2, 3.—Diagrams of Case 11, Table II.   Left pneumothorax;
intra-pleural pressure = 7 inches of water.

Diag. 2, taken during life.—*a.* Outline of heart.   *b.* Position of
undulating impulse.   *c.* Apex beat.   *e.* Some dulness and moist crack-
ling rhonchus.   Left side enlarged and hyperresonant; no respiratory
sound save at *d,* where inspiration has some hollow quality.   Diag. 3
indicates exact position of parts as found *post mortem.*   *a.* Tube
inserted into left pleura to ascertain intra-thoracic pressure.   The
shading at the upper part on this side indicates the compressed left
lung.

The following diagrams illustrate these three cases.   It
will be observed from the remarks attached to two of these
cases in the Table that a small quantity of purulent fluid
was found in the pleura of one and about a pint of puru-
lent fluid in that of the other.   It must, however, I think,
be granted that this amount of fluid was quite insufficient
to account for any appreciable displacement, especially
when it is remembered that it did not cause any compres-
sion of the air within the pleura.   In the third case there
was no fluid present.

Some seven or eight years ago I endeavoured to explain
and to illustrate the essential cause of the cardiac displace-
ment in pneumothorax by some simple experiments which
were related to this Society.[1]   I need only briefly mention
them now.

[1] 'Medical Times and Gazette,' Jan. and Feb., 1869.

FIG. 4.   FIG. 5.

Figs. 4, 5.—Case No. 9, Table II. Left pneumothorax; no intra-thoracic pressure.

Diag. 4.—Percussion limits marked out before opening thorax. *a, b.* Boundary of left pleura. *c.* Displaced cardiac dulness. Diag. 5.—Exact position of heart ascertained on removing sternum. A small puncture is indicated at fourth space, through which a stilette was thrust to fix the heart *in sitû* before removing sternum.

FIG. 6.   FIG. 7.

Figs. 6, 7.—Case No. 10, Table II. Left pneumothorax; no intra-thoracic pressure.

Diag. 6, taken before removing sternum, showing displaced cardiac dulness. —*st.* Point at which stilette inserted to transfix heart *in sitû.* *a, b.* Margin of left pleural cavity. Diag. 7 shows position of heart and collapsed left lung as seen on removing cartilages.

NOTE.—The roughness of the above diagrams is due to their being exact copies, on a reduced scale, from my clinical and post-mortem note-books.

FIG. 8.

Case No. 17, Table II. Left pneumothorax; no intra-thoracic pressure. R. Collapsed left lung, communicating with pleura by free opening at a.

1. In the dead subject, the chest being healthy, a long and light needle was thrust vertically into the heart to serve as an index (vide woodcuts Nos. 9 and 10), and on the left pleura being then cautiously opened so as to freely admit air, the needle became slightly deflected, so as to indicate a movement of the heart towards the right. An inspiration was now imitated by evenly raising both arms above the head, and the displacement became more marked.

2. A similar experiment was made upon a living dog whilst under chloroform. The deflection of the needle was still more marked.

The inference from these experiments was that the elastic tension of one lung, when unopposed by that of the other, was sufficient to draw aside the mediastinum and with it the heart. In the case of the dog the displacement was still more marked, because the lungs being in action their tension was naturally increased with inspiration.

The chief conclusions which seem to follow from the facts recorded in the table are—

1. That displacement of the heart is an immediate and a most important sign of pneumothorax, depending

Fig. 9.
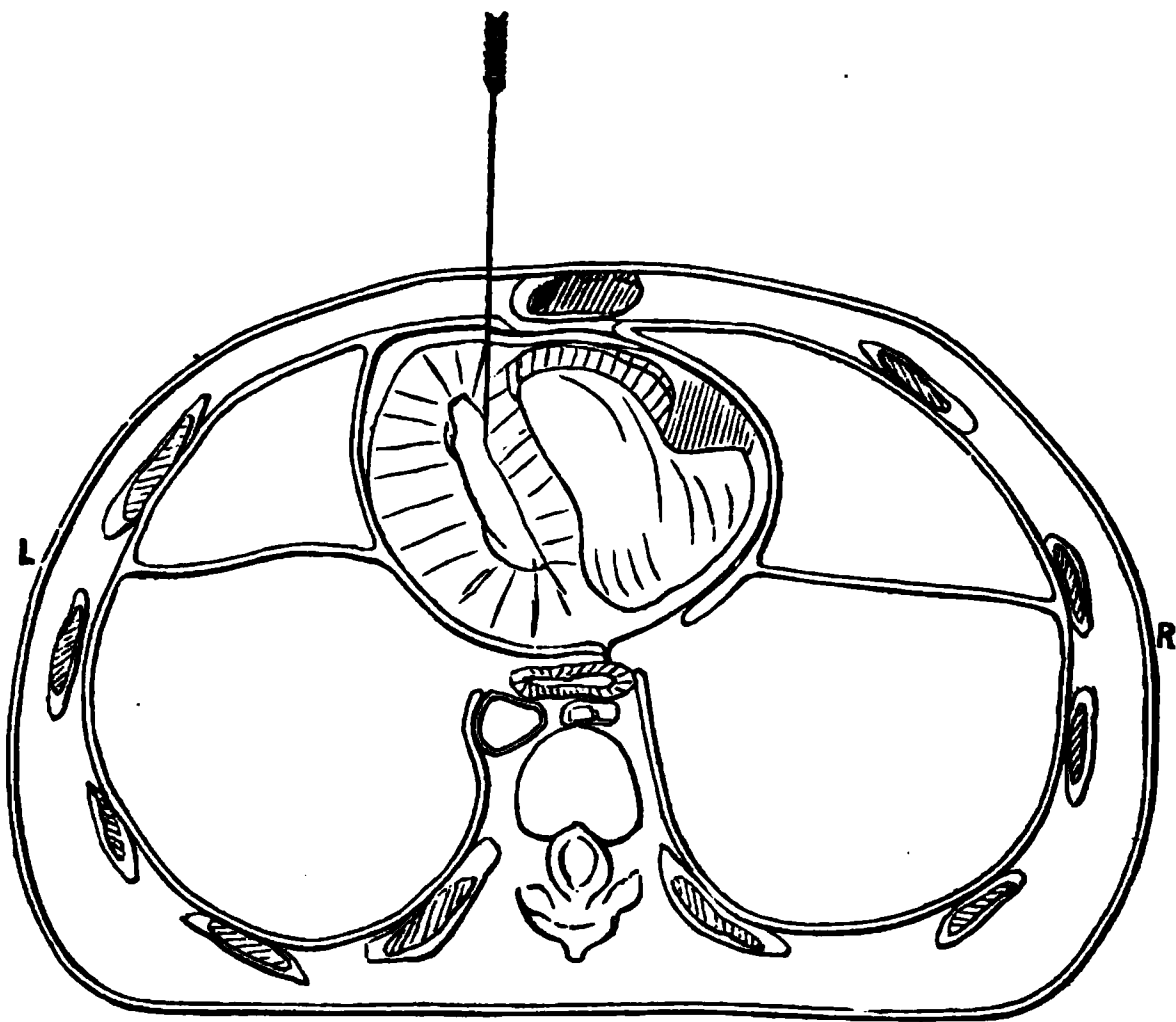
Fig. 10.
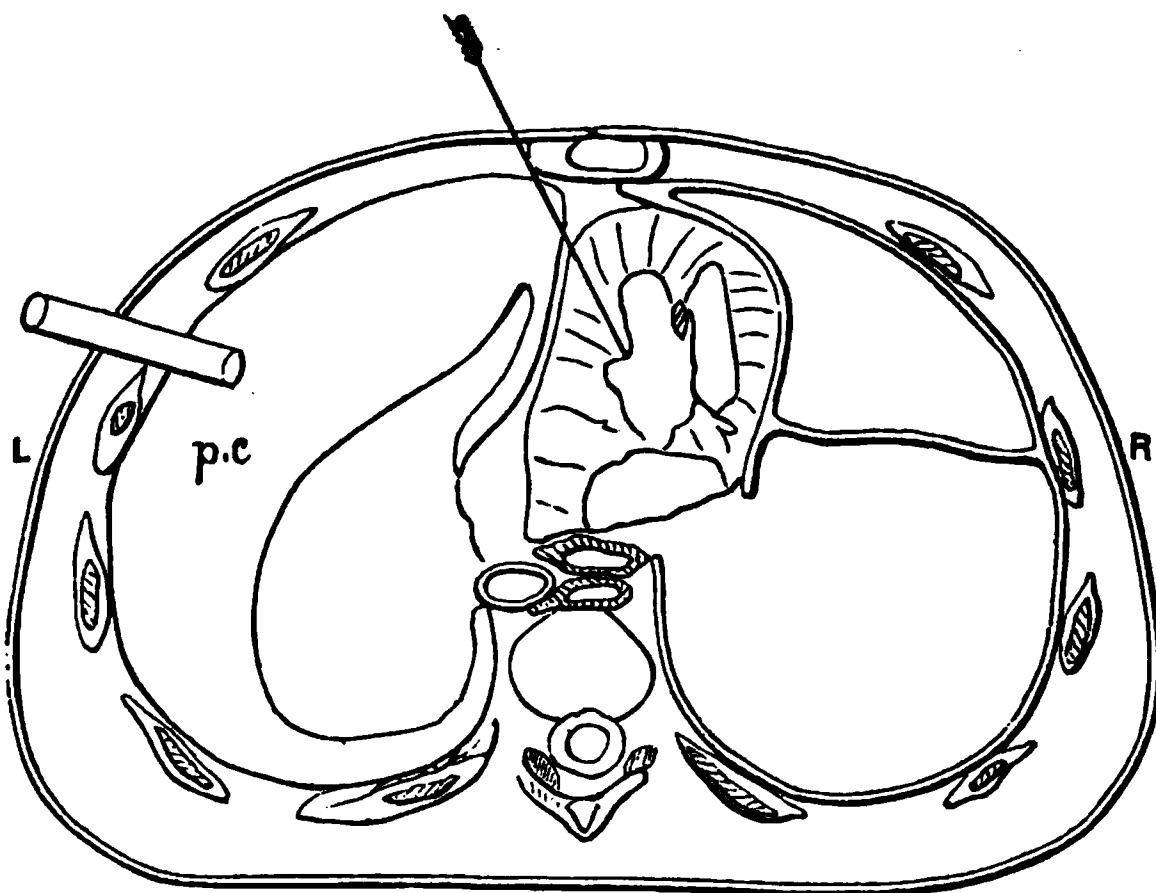
Figs. 9, 10.—Diagrams of sections of the chest to illustrate experiments showing the effect upon the position of the heart of admitting air into one pleural cavity. Fig. 10 shows deflection of an arrow thrust vertically into the heart on making a free communication between the left pleural cavity (pc) and the external air. (Adapted from Pirogoff's Anatomical Plates.)

upon the mere presence of air in the pleura and upon the contractility of the unruptured lung.

2. That the cardiac displacement is by no means necessarily a sign of intra-pleural pressure, since, as will be seen on glancing at the diagrams of Cases 9 and 10 (*vide* woodcuts Nos. 4 to 7), the heart may be displaced to the right of the sternum without there being any pressure.

3. Hence, in discussing the question of paracentesis in any given case of pneumothorax we must take into consideration other things besides the position of the heart.

The above views are, however, by no means generally accepted even by those who are acquainted with them. Dr. Hayden, of Dublin, in his recent elaborate work on 'Diseases of the Heart,' at p. 100, after very fully and fairly stating my experiments and deductions, gives what he conceives to be good reasons for adhering to the doctrine of lateral displacement of the heart by pressure, "as promulgated by Bertin, Laennec, Hope, Stokes, and Walshe, and almost universally held at the present time." In the first place, Dr. Hayden thinks that in my experiments the deflection of the needle was "directly and exclusively due to pressure upon the mediastinum of the air admitted into the pleura. This air being free, and its pressure therefore not being neutralised by the elastic reaction of the lung, as was the case with that included within the pulmonary tissue of the opposite lung." If Dr. Hayden's objections be valid they will not fail to be ably urged by some of the Fellows present, and it would be taking up too much of the Society's time to refer at length to his arguments now. I must say, however, that it is difficult to understand how there can be any difference between the atmospheric pressure within the lung and that outside the thorax; and the atmospheric pressure being the same, therefore, on both sides of the balance, but the elastic tension on the one side being annulled, the elastic tension on the other side seems to be the only force which, thus unopposed, can rightly be considered as disturbing the equilibrium. I would beg further to remark respecting

two points in Dr. Hayden's experiment undertaken in order to test the accuracy of my views. Firstly, that he apparently does not consider that the thoracic wall has anything to do with the question ; and, secondly, he seems to think that by inflating the lung he can imitate its normal expansion. Thus, Dr. Hayden[1] commences his experiment by removing the anterior wall of the chest of a subject brought in for dissection, except the sternum, which he divides in the median line with a saw. He then detaches the root of the left lung and corks up its main bronchus. He then further proceeds to inflate the right lung by blowing into the trachea to ascertain the effect upon the heart's position. Is it possible to conceive any experiment performed under conditions more diverse from those which are natural ? Yet it is from such an experiment that Dr. Hayden draws conclusions favorable to the views of the distinguished observers already quoted.

Whilst contending that the *primary* cause of displacement of the heart in pneumothorax is not air-pressure acting from the diseased side, but lung-traction acting upon the healthy side, I am, of course, fully aware that such displacement must necessarily be increased should air accumulate in the pleura.

The mechanism of displacement of the heart in cases of fluid effusion into the pleura is essentially the same, but the occupation of the pleura by the effusion being gradual instead of sudden, as is its occupation by air, the displacement of the heart is slow instead of being instantaneous.

Cardiac dislocation occurs in pleurisy *pari passu* with the effusion, whereas if its occurrence were a matter of *pressure* it should not take place so long as the fluid no more than occupied the space left by the contracted lung.

I would mention, by the way, one little point of clinical interest to be observed in cases of moderate effusion into the right pleura, viz. that in such cases the heart may appear to beat more to the right than natural. This is

[1] P. 102.

due to the right lung as it collapses uncovering the right side of the heart, the impulse of which is much more readily transmitted through fluid than through the spongy lung. Thus, whilst the heart is really displaced slightly to the left, it may seem not to be displaced at all or even to be somewhat more to the right than natural.

In cases of considerable fluid effusion into the pleura there is, of course, always pressure upon the heart. I have measured this intra-pleural pressure in several cases at the various periods of removal of the fluid by thoracentesis, and have found the fluid pressure to vary from half an inch to an inch and a half of *mercury* at the moment of puncturing the chest. I subjoin a table giving particulars of three such cases, but need not further refer to them.

It may, however, at first appear somewhat surprising that with the considerable intra-pleural pressure here recorded there is not more difference between the chest measurements on the two sides in cases of effusion into the pleura. Sometimes these measurements are equal; there is rarely an inch or an inch and a half of difference between them. A little consideration, however, points out the reason of this, and it is the last matter I have to touch upon. When an effusion, whether of air or fluid, takes place into the pleura on one side, in proportion as the mediastinum encroaches upon the healthy side the thoracic parietes on that side expand in consequence of the relaxed lung setting free their excentric resilience. This tends to equalise the measurements of the two sides, but to increase the total circumference of the chest.

Note.—I have only been able briefly to glance at the elaborate work just published by Dr. Ransome on stethometry. As in his paper referred to, at page 170, however, the thoracic movements recorded in Dr. Ransome's present tables appear to be those of forced breathing, with which the observations in the present paper do not deal. At page 96 Dr. Ransome makes an interesting observation with reference to forced breathing, which, as I have shown, should also (proportionately) hold good with ordinary respiration, viz. that in measuring the movements of forced breathing by means of his three-planed stethometer, " the index of the *forward* dial generally moves first, a forward push of 0·25 to 0·30 inch being often noticed before any decided action of the other indices takes place. . . . This fact," observes Dr. Ransome, " would probably be sufficient to prove that there must be some preliminary indrawing of the chest-wall in the expiratory act."

TABLE III.—*Showing intra-pleural pressure in cases of empyema.*

Case.	Sex.	Age.	Side.	Duration.	Intra-pleural pressure (mercury).	Fluid removed.	Chest measurements before operation.
T. W.	M.	30	Left	10 months	Movement Commencement of operation + 1 in. ... ⅛th in. Termination of operation + ¼ in. ... ⅜th in.	Thin pus, 4½ pints	Left semicircumference ... 18½ in. Right ,, ... 18¼ in. Difference ... ... ¼
T. B.	M.	28	Right	5 years	Commencement of operation + ⅜ in. Termination of operation — 1 in.	Pus, 4 pints	Right semicircumference... 18¼ in. Left ,, ... 17⅜ in. Difference ... ... ⅞ in.
E. M.	M.	22	Left	...	Commencement of operation— During inspiration + 1¼ in. During expiration + 1¼ in. } Movement ¼ in. Termination of operation ± level	Pus, 4¾ pints	Left semicircumference ... 17⅜ in. Right ,, ... 16⅜ in. Difference ... ... 1¼ in.

ON SOME OF THE

HISTOLOGICAL CHANGES

FOUND IN

CANCER OF THE SKIN OR EPITHELIOMA,

WITH

SPECIAL REFERENCE TO THE SOURCE OF THE NEWLY FORMED EPITHELIAL CELLS.

BY

GEORGE THIN, M.D.

THE opinions entertained by pathologists regarding the origin of the cells which form the epithelial growths that constitute one of the main features in cancer of the skin may be classified under the following heads :

Virchow and those who follow him believe that the epithelium which constitutes the new and morbid growth is formed by the cellular elements of the subjacent connective tissue.

Others, with Thiersch and Waldeyer, deny this participation of the subjacent connective tissue, and believe that the epithelial cells of the new growth are produced exclusively by proliferation of the pre-existing epithelial cells of the rete mucosum, glands, and hair-follicles.

Most of the best known names in pathology may be

ranged on the side of one or other of these two views, although there is considerable difference of opinion regarding the manner in which the connective tissue on the one hand or the epithelium on the other is supposed to produce the new cells.

Exceptional views have been entertained by Köster, who believes that the new growth takes its origin in a proliferation of the epithelium of the lymphatic vessels, and by Classen, who in a case of cancer of the eyeball extending to the cornea convinced himself that the new cells were derived from the lymph-corpuscles.

The opinions which I shall develop in this memoir were formed after I had examined cancerous tumours from various parts of the body, and more especially after careful study of the appearances found in epitheliomata of the lip.

The preparations which are more particularly described, and from which the drawings have been made, were obtained from a case of epithelioma of the lip of a woman, diagnosed and operated on by Mr. Bell, of the Edinburgh Royal Infirmary.

I am indebted to Mr. Bell for the following notes of the case, which he has kindly sent to me at my request :

B. W—, æt. 67, from Penicuik, was the case from which I removed the specimen of epithelioma I sent to you for examination. She was a thin, somewhat withered old woman, who lived alone and supported herself by sewing. The malady was an exceedingly typical example of epithelioma, and had the usual history of the appearance, first of a small hard nodule, which broke, leaving an ulcer with a hard base, on which successive scabs formed and fell, leaving a steadily growing painful and fœtid ulcer. The pipe which she owned to using frequently was a very short and dirty black cutty. The glands were not affected. I removed it by a V-shaped incision, and, as usual, with a rapid recovery. Such cases are exceedingly common in the male sex, and this was as perfect and typical a specimen as could be got. I am quite familiar

with it in the female sex, having now operated seven times on old women, all of whom were in the constant habit of smoking dirty, short, hot pipes. I have never seen epithelioma in a female who did *not* smoke.

I enclose you a copy of the case as extracted from the hospital books.

Extract from case-book.

B. W—, æt. 67, a sewing woman, residing in Bread Street, Penicuik, was admitted into Ward 1, surgical, suffering from epithelioma of the lower lip, on January 28th, 1875.

History.—About a year previous to admission the patient observed a small hard point in the lower lip close to the left angle of the mouth. About two months previous to admission the surface broke and an ulcer appeared, having a hard base and uneven edges. The patient stated that she had been accustomed to smoke, the pipe used being generally a short clay.

Appearance on admission.—A small ulcer existed at the situation above mentioned. It extended through the whole thickness of the lip, involving both skin and mucous membrane. The hardness did not extend far. The glands situated below the jaw and in the neck were not at all affected.

Treatment.—On the 30th January, 1875, Dr. Bell performed the following operation :

The epithelioma was removed by a V-shaped incision ; what arteries bled were not ligatured, but twisted. The edges of mucous membrane and skin were carefully brought together, the former by silk sutures and the latter by silver wire. During the evening after operation troublesome hæmorrhage supervened, and the house-surgeon, Dr. Cotton, was compelled to undo the stitches. The hæmorrhage was of the nature of a general oozing, which was in the end stopped by the use of Argentic. nitrate. Turpentine was administered internally in capsules.

In the course of the afternoon of the 31st Dr. Bell put in the stitches again. At this time the patient's pulse was 100, and very weak.

February 1st.—Pulse stronger ; reaction set in.

3rd.—As the edges of the wound showed a tendency to eversion, Dr. Bell put in a harelip needle.

9th.—Patient is getting on very nicely. The harelip needle was removed to-day.

11th.—Patient was to-day *discharged.*

Result.—Cured.

I found that the knowledge which can be obtained by examining sections of cancerous tumours depends very much on the mode of treatment to which the excised tumour is subjected. Profiting by this experience I treated the specimens which furnished the preparations, which I shall now describe, in the following manner :

Thin pieces were put in a one eighth per cent. solution of chromic acid, and the solution was changed daily for several days. They were then put in a quarter per cent. solution for two days, and finally in a half per cent. solution for four or five days.

The pieces thus hardened were embedded in wax and oil, and sections sufficiently thin for examination could be made. To obtain very thin sections subsequent hardening in methylated alcohol for twenty-four hours is advisable. But as a rule the less the specimen is brought into contact with alcohol the better. . In sections cut directly after the action of the weak chromic acid, the abundant cell infiltration in the neighbourhood of the epithelial growth can be advantageously examined, the individual cells being well preserved.

If the sections are allowed to macerate for one or two days in distilled water they part _with a portion of the chromic acid, and are then fitted for staining, solution of logwood acting readily on nuclei of cells so treated. The preparations should be examined in glycerine, as they suffer from the action of the alcohol and turpentine or oil

of cloves which are necessary if Canada balsam or Dammar varnish is used.

I attach importance to these details because I have by following them obtained better preparations than when the tissues were hardened by stronger solutions of chromic acid and by alcohol.

Another series of preparations, and amongst them those to which I assign most importance, was obtained by the action of solution of osmic acid.

As the use of osmic acid in histological studies is in this country still comparatively limited, I may be pardoned a few preliminary remarks regarding the kind of effects which can be obtained by subjecting the fresh tissues to its action, as it will be necessary to bear these in mind in connection with the appearances I have to describe in the cancer preparations.

Osmic acid solution rapidly stains fat and medullated nerve-fibres black, but with its valuable uses in this respect I have at present nothing further to do. It stains epithelial cells a varying shade of brownish yellow, fibrillary tissue is faintly coloured, and elastic fibres not at all. Lymph-cells acquire a faint dull hue, and are seen as granular bodies. The red blood-corpuscles are seen as rounded or quadrangular elements, in which the dark staining produced by the osmic acid is mingled with a slight tinge of their original colour. But distinct from the staining effects of the solution there is a peculiar " fixing " of all the tissue-elements.

Unlike many other hardening and staining reagents, it does not produce shrinking, and the most delicate structures are by its use preserved in their natural size and relations.

To understand the advantages which result from the application of osmic acid to the study of the histology of cancerous growths, it is only necessary to consider what has been already stated generally regarding its effects.

It stains the epithelium and the fibrillary tissue, fixes the free cells, makes the red blood-corpuscles prominent,

enables us to identify colourless blood-corpuscles or lymph-cells, and indicates gaps and spaces. It further hardens sufficiently to enable sections to be made without the use of alcohol.

I have not hitherto found that it stained the fixed cells of the connective tissue in cancerous skin, but as it has produced this result in other organs a similar effect in skin is probably only a question of successful manipulation. Immediately after Mr. Bell had excised the affected portion of the lip, thin slices through its whole thickness were placed in ¼ per cent. solution of osmic acid and sent me by post. I embedded portions of the slices the following day and prepared sections which were variously stained and mounted, sometimes in glycerine and sometimes in, what is better, a saturated solution of acetate of potash.

The results obtained by careful hardening in weak chromic acid were confirmed by the osmic acid sections, and appearances became visible which I had not detected by other methods.

The changes which the epithelial cells undergo in can-cerous skin have been frequently described. I shall dis-cuss separately the changes I observed in the cell and the nucleus. Like other observers I distinguish two kinds of change in the cell. In the centre of the enlarged rete mucosum and in the concentric layers of the laminated capsules the cells are seen to be much larger than in the normal tissue. They flatten out, sometimes become ex-ceedingly long, twice or three times the length of an epidermic cell in healthy skin, and acquire a dried horny aspect. Others swell and have the appearance of a some-what spherical, homogeneous, vitreous mass. One or more such cells may be often seen occupying the centre of a lami-nated capsule. I have satisfied myself that a laminated capsule frequently has its point of departure in an altered duct of one of the skin-glands, and I have found changes in the cells of little altered and recognisable sweat-glands, which represent gradations between normal cells and the large vitreous cells in the centre of the laminated capsules.

This is not, however, invariably the case. A cell of the rete mucosum sometimes undergoes the peculiar degeneration which indicates the first point of departure.

The cells of the sebaceous glands undergo changes in which the influence of their original peculiarities can be recognised, the waxy, somewhat glistening appearance of these cells being still distinctly visible. In a section which contains a great number of the cylindrical and pear-shaped epidermic masses of cancerous skin it is often possible to distinguish the growths that have taken origin from the sweat- and sebaceous glands respectively by the peculiar appearances presented by the central cells. Growths, on the other hand, that spring from the inter-papillary projections of the rete mucosum uniformly retain the usual characteristic aspect of the cells of that layer.

The cells that form the external layers of the deep projecting epithelial masses that can be recognised as enlarged sweat-glands and ducts have not the appearances which I attribute to glandular origin, but on the contrary have nothing to distinguish them from the cells which are continuous with the rete.

The cells in the centre of the epithelial masses are, as a rule, larger thant hose on the borders, and in thin sections the serrate edges, which have given the designation "prickle cells" to epithelium on which they are seen, are more distinct than in normal epithelium. But they appear to be in no way connected with the cancerous development, which simply admits of their being more easily detected.

I can confirm Thiersch as against Köster, that the epithelial growth takes place from the rete mucosum, glands, and hair-follicles.

I have not once seen an epithelial cell constricted or marked by lines in any way whatever that would indicate that it was in process of dividing into two, or giving birth to an endogenous progeny, and after a careful examination of a great number of preparations I have come to the conclusion that an epithelial cell in a cancerous tissue, although it undergoes various changes, never begets

another cell or cells. The idea of proliferation has originated, as I believe, in an erroneous interpretation given to certain changes that take place in the nucleus, which I shall now describe, and to other appearances seen in the *débris* of degenerated cells and nuclei, which I shall notice further on.

The nuclei of the epithelial cells in cancer may undergo a change similar to that which distinguishes the nuclei of the horny layer of the epidermis. They appear to dry and wither, and in the manipulation of the tissue they are apt to fall out of the cell, and instead of a nucleus a nuclear vacuole is left. In other cases the nucleus is suspended in the cell as an apparently dried, delicate, membranous substance, in the centre of which a large nucleolus is seen. The impression produced on the observer is as if the fluid contents of a vesicle had disappeared and had left behind an envelope and a network of exceedingly fine threads, in the centre of which the nucleolus is supported. The nucleus in this case is not larger than it is in the normal cell, but the nucleolus, if not actually larger, appears to be so, and I am disposed to believe that it is actually larger. At all events it is certainly altered in consistence and colour. It is darker than normal, and free nucleoli may be seen amongst the cells nearly as large as red blood-corpuscles, and having evidently a power of resistance which they do not possess in normal tissues. To judge by their resemblance in colour and appearance to the substance of the horny looking, enlarged, flattened cells, it would seem as if the change they had undergone was of a similar nature—that they had also undergone a horny degeneration.

Sometimes the withered nucleus can be seen to be divided by delicate radiating threads into four compartments, and in each compartment there is a small nucleolus. The aggregate bulk of the four nucleoli does not exceed that of the single enlarged nucleolus which I have just described. In this case the size of the entire nuclear space is neither enlarged nor constricted. The presence of

several nucleoli in the nucleus may seem to justify the idea that the nucleus divides, but there is no evidence that there is any connection between the two processes. The more numerous the nucleoli are the smaller they are, from which I infer that they are a product of a division which is nothing more than a disintegration. I believe, for reasons into which I need not enter further here, as the question is not an essential one in this connection, that the disintegration is a separation of elements which were originally isolated, and which were welded together when the cell assumed its permanent form.

A mode of division of the nucleus which is, as far as I have observed, rare, and the best-marked instances of which I have observed in preparations of scirrhus of the breast, is when, instead of one large nucleus, the cell contains several smaller rounded bodies, which are not bounded by a distinctly demarcated, vesicular, or nuclear space. Each of these bodies consists of a central point and a homogeneous border. They are smaller than any cellular element ever seen, either free, as forming part of the so-called cell-infiltration, or as forming one of the cells of the cancerous epithelium, and possess a characteristic appearance which, irrespective of size, would prevent their being confounded with anything else. I believe them to be products of disintegration of a nucleus which has swelled before going to pieces, and that the envelope which separates the nucleus from the other contents of the cell being ruptured, the nucleolar masses spread themselves through the substance of the cell. The appearance is, besides, sufficiently rare to be left out of account in a consideration of the question of cell growth.

In cancer the nucleus of the epithelial cell undergoes one of two transformations. It withers and falls into pieces, or in a swollen vitreous cell it undergoes the characteristic change that takes place in the cell-substance, but to a less degree apparently.

After the cell and nucleus have undergone further retrograde changes in addition to those which I have de-

scribed, another appearance is produced which is note-worthy, as being apt to lead to a mistaken idea regarding the growth of new cells. The original cell may have undergone molecular disintegration until there is nothing left of it but an uneven ring or ridge, which adheres to the adjacent tissue and corresponds to the contour of the cell. The rest of the cell and the nucleus have disappeared. I have seen this modified in so far as the cell was represented by two concentric ridges, an outer one corresponding to the · contour of the cell, and an inner one to the circumference of the nucleus. Within these rings or ridges a lymph-corpuscle often fixes itself and, as we shall afterwards see, undergoes structural changes. It is then apt to be regarded as a product of the dead cell if the appearance is not carefully controlled by appropriate methods.

Thus, all the changes I have observed in the nucleus and cell are connected with death and disintegration. Of reproduction there is no evidence.

Surrounding the growing epithelial patches there is invariably to be observed a greater or less number of cells which are not seen in the normal tissue, and to these cells I now direct attention. They vary in size from that of the smallest lymph-corpuscles to that of an average red blood-corpuscle of the frog. Few of them attain the latter size. The smaller ones are rounded, but when seen in osmic acid preparations are frequently compressed laterally. The larger ones are mostly polygonal, a form evidently produced by the pressure they mutually exert on each other. The further we go from the epithelial growth the smaller the cells become, the very gradual transition stages by which the larger epithelial-looking cells pass into the small rounded corpuscles being almost imperceptible. In order to understand the disposition of these cells in the corium and subcutaneous tissue it is necessary to leave them for a little and consider the changes that take place in the fibrillary tissue in skin which is the seat of cancerous growth. And, first, one word in regard to

the anatomy of that tissue. The fibrillary or gelatinous
substance of the skin is composed of bands or bundles,
and as the nomenclature which has been applied to the
forms which these assume does not appear to me to
be exact, it is necessary that I should define the terms
I shall use. I recognise in the fibrillary tissue of
the skin as a primary element, a definite, even, some-
what flattened, cylindrical body of uniform calibre, the
breadth approaching approximatively the diameter of a
human red blood-corpuscle. When the cutis has been
hardened in spirit all traces of the unity of structure
of this band usually disappear, and portions of it split
longitudinally are seen. The term white fibre has been
applied indiscriminately to these longitudinal fractions of
the bundle and to the bundle itself when seen entire.
For this reason I do not use the term in case it should
lead to a misapprehension of my meaning. For a similar
reason I reject the use of the word fibre as applied to this
tissue. The term fibrilla is sometimes used in reference
to these bundles and their fragments, being applied in the
same way as the term white fibre. I reserve the use
of the term fibrilla for a different element. It is possible
by maceration in blood serum or osmic acid, and with
most certainty in the former, to resolve tendon into a
multitude of exceedingly delicate threads or filaments.
With more difficulty I have succeeded in demonstrating
the same extremely fine filaments in the fibrillary tissue of
the skin, and to them I think the term fibrilla may be
most appropriately applied. The next definite element
is the cylindrical band already mentioned, and for con-
venience I shall in this paper designate it a primary
bundle. Several of these applied to each other longi-
tudinally form the next definite structure, and I shall call
it a secondary bundle. Large groups of such secondary
bundles can often be distinguished in skin preparations,
and may be designated tertiary bundles. Between these
tertiary bundles ramify the larger blood - vessels and
nerves.

This nomenclature is not defended as the best that can be used, but it will serve until a better is selected, and the older terms are not sufficiently exact for my purpose.

In the skin of the dog Dr. Stirling ('Journal of Anatomy and Physiology,' 1876) has been able to isolate by artificial digestion sheaths which invest what he terms the "larger and very fine," what I should call the secondary and primary, bundles. In the human skin I have been able to fill the spaces between the tertiary bundles with metallic deposit, which indicated the presence in the spaces of a considerable quantity of lymph fluid. Without entering further into histological details I may state once for all that a constant circulation of lymph fluid between the primary bundles, between the secondary bundles, and between the tertiary bundles, may be fairly assumed. The lymph system of these spaces must not be confounded with the lymphatic vessels properly so called, and of which they are in the position of radicles. In the lymph fluid between the bundles a certain number of colourless blood-corpuscles (or lymph-corpuscles) are found normally. In inflammation and in other pathological conditions their number is greatly increased.

The fibrillary tissue in cancer of the skin is seen to be modified in two degrees. In the narrow papillæ compressed on both sides by the epithelial growth, and deeper in the corium when the growing epithelial masses approach each other, the arrangement in bundles is lost, and the tissue is represented by scant irregular shreds. It has to a great extent disappeared. These shreds of tissue often contain numbers of red blood-corpuscles in their meshes, and capillary vessels can often be detected amongst them in osmic acid preparations by the presence of the red blood-corpuscles in unbroken lines.

Farther removed from the epithelial growth the arrangement in primary bundles is still intact, but the substance proper of the bundles has been to a great extent absorbed. The contour of the bundles is indicated by straight,

unbroken, narrow lines of a highly refractive substance. Farther removed from the epithelial growth the condition of the bundles is normal.

The arrangement of the new cells to the fibrillary tissue will now be more easily understood. In the narrow clefts between the encroaching epithelium the shreds of the disorganized bundles are infiltrated with cells. As the relative arrangement of the cells is no longer determined by the fibrillary tissue, they are seen in sections either grouped in irregular masses or presenting the more or less regular appearance of a layer. In the adjacent parts, where the fibrillary tissue has been partly absorbed, but still retains its arrangement, the cells can be seen disposed in rows between the primary bundles or lying on them, their disposition being accurately in accordance with the anatomical relations of the bundles. When the accidental effect of the knife in cutting the section has been to isolate a secondary bundle more or less completely, it can be sometimes seen to be encrusted with the cells. The wide, frequently pyramidal-shaped, spaces between the tertiary bundles are often filled by masses of them.

But the cell infiltration does not stop here. In the surrounding fibrillary tissue, where the appearance and arrangement of the bundles are absolutely normal, single rows of cells can often be seen between the bundles, being a continuation of the same infiltration that is so abundantly present in the parts nearer the cancerous growth. The cell infiltration of cancerous skin extends, therefore, from the parts which are profoundly affected to the adjacent tissues, which are in other respects still apparently healthy.

The imperfect knowledge which we possess of the development and nature of the cellular elements of connective tissue renders an inquiry into the source of cell infiltration, such as that present in cancer of the skin, a very difficult task. Their development from the epithelium of the rete or the glands, in spite of the most elaborate studies by many competent observers, remains in the present state of science a bare hypothesis, unsupported by

any evidence stronger than the weight that is attributed to the division of the nucleus into several pieces. There has never been any satisfactory evidence adduced that this division of the nucleus is connected with a development of new cells. In the cancerous process I have found that it is simply a stage in the cell disintegration, and in all the sections I have examined I have never seen any appearance that has indicated a development either by the process which is understood by the term proliferation or by endogenous cell growth. The appearances actually observed have been already described in detail, and I believe they support me in denying that the source of the new cells is to be found in the pre-existing epithelium. In the present state of opinion regarding the histology of the skin the question seems to me to be limited to the consideration of one of two sources, and we have to decide whether these cells originate from cells already existing in the tissue or are derived by a process of extravasation from the blood-vessels. I presume that their origin from a blastema will hardly be considered a possible one.

I have now to consider whether the new growth may not spring by some process of what is called " proliferation" from the cells of the connective tissue. Here, however, we are at once met by the difficulty that we know very little indeed about these cells. That a great number of cells exist is evident from the number of nuclei that can be stained by certain modes of preparation. Biesiadecki states that there are spindle (or fusiform) cells in the human corium, and this fact appears to me to have been demonstrated by Dr. Stirling for the skin of the dog. Dr. Stirling's plates show further that there are large round nuclei in the skin, distinct from the spindle elements, but he leaves the nature of the cells to which these nuclei belong undetermined. As the result of researches which I have made on the cornea, to which I cannot do more than allude here, and of what is now established as regards tendon, I believe it is fair to assume that, in accordance with the similarity

of structure, there must exist in the corium a system of flat cells applied to the surface of the bundles of fibrillary tissue. And, in point of fact, I have isolated from the cutis of the frog and the ox by warm saturated solution of caustic potash[1] flat cells similar to those which by the same process I had isolated from the cornea. As far as I know, these cells have never been seen by any other process, but cells exactly similar have been isolated by Ranvier[2] from the subcutaneous tissue into which a weak solution of nitrate of silver had been injected. The nuclei figured by Dr. Stirling seem to me to belong to the same cells, although it is right to mention that he regards them as belonging to lymph-corpuscles.

I have by means of several methods satisfied myself that there are stellate cells in the corium. The cell-protoplasm is very scant, and the fibres are straight and glistening, and do not stain in carmine or logwood. The plasmatic cell of Virchow is not a cell, but is simply a space between the bundles.

Thus, I believe that there exist in the skin stellate, spindle, and flat cells; but I hasten to remark that whether my views in this respect are for the present received or not, my general conclusions regarding the source of the cell infiltration remain unaffected, as my observations, which relate to the cells of the corium, are as regards this particular question entirely negative.

The existence of numerous stellate and spindle cells in the parts of the corium adjacent to the epithelial cancerous

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[1] For further details regarding this process see a memoir on hyaline cartilage in the 'Quart. Journ. Mic. Science,' January, 1876.

[2] 'Archives de Physiologie,' 1869. The exact bearing of Ranvier's discovery has been obscured by several subsequent investigators, who have described isolated nuclei adherent to torn membranous substance as flat cells with fine processes. This error is due to unsuccessful manipulation. The flat cells of the subcutaneous tissue have no processes. They are easily demonstrated by injecting a weak solution of nitrate of silver under the skin of a mouse's back. It can then be seen further that they are not isolated, but that they form layers, such as that figured by me in the 'Proceedings of the Royal Society,' No. 155, 1874, figure 18, pl. ix.

growth is indicated by minute fatty globules embedded in a granular substance which is seen in the spindle and in the stellate forms. These are prominent in osmic acid preparations, the fatty globules being stained black. This appearance is seen occasionally in cancerous skin, but I have met with it more frequently and much more abundantly in scirrhus mammæ. In the breast I have been able to follow the degenerating protoplasm into fine, unstained processes, which demonstrated the nature of the cell. The distinction between the spindle cells and the cancerous epithelium was well marked.

No production of anything like new cells of any kind was ever found by me as taking place in these cells.

In thin sections of cancerous skin hardened in chromic acid or spirit and stained in logwood, minute stellate and spindle cells can be seen between the epithelium of the new growth. Little more than the nucleus is visible, but the beginning of the fine processes can generally be seen. The nucleus of the stellate cell is pyramidal and that of the spindle cell narrow and elongated. It is impossible to confound them with the epithelial cells, in whatever stage of growth or degeneration these may be. There is no transition between the minute, deeply stained nucleus of the branched cell and the large, round, faintly stained nucleus with central nucleolus of the epithelial cell. From the position and number of the nuclei and from what is visible of the processes it is evident that there is a highly developed interepithelial system of stellate and spindle cells in the epithelial growth of cancer. Similar nuclei and processes can be observed in the epithelium of healthy skin, although their demonstration is more difficult and succeeds to a less extent when the ordinary available methods are followed.

These preparations confirm the conclusion I have drawn from the osmic acid preparations—that the development of the epithelium is independent of the branched cells of the tissue.

In osmic acid preparations of cancerous skin large oval

nuclei can be sometimes observed on thin membranous prolongations from the border of the rete, on the surface of isolated bundles, and in the walls of blood-vessels. These nuclei are faintly marked, show no signs of division, ·and nothing is seen of the cell to which they belong. They are distinct from any form of epithelial degeneration observed and from the lymph-cells. The nucleus is indeed larger than many of the latter. I have never observed these nuclei in osmic acid preparations of the healthy skin. I do not on this account connect their presence with cancer, but believe them to be the nuclei of the flat cells which are shown to exist in the skin by means of other processes. I have not observed them in preparations made from cancerous skin hardened by any other reagent than osmic acid. I infer that their appearance in epithelioma, whilst they are not seen in the healthy corium which has been subjected to the action of the same solution, is probably due to a change in the condition of the nucleus itself. At the same time it is not to be left out of account that the widening of the interstices of the tissue as a consequence of the morbid process may have resulted in the " fixing " of the nuclei by affording easy access to the entrance of the osmic acid.

It is important in considering the question of the possible development of cancerous cells from the flat cells of the corium to bear in mind that the nuclei which evidently belong to these cells are found unbroken and undivided, even when they are surrounded by cells of new formation. Amongst the drawings which accompany this paper is one (Fig. 2) which shows a secondary bundle with small cells clustering round it, and amongst these the oval nuclei on its surface are seen to be entire.

As far as it is possible to obtain evidence regarding the behaviour of cells of which we know so little as the cells of the corium, the presumption is strong that they have no share in the production of the new cells, and there is not a single fact known that suggests that they have anything to do with it. From the epithelial form and appearance

which is characteristic of flat cells, it seemed to me at one time that the epithelial cells of the cancerous growth might possibly be due to some changed condition in these cells by which they attained the more advanced horny development of the epidermic cells. Of this, however, I have found no evidence, and the hypothesis is, in my opinion, excluded by the positive evidence that the cells are derived from another source, which I shall now indicate.

The cells which are found in rows between the apparently unaffected bundles of fibrillary tissue and the most of those between the bundles that are partly absorbed but still retain their form and arrangement are, in their microscopic appearances, indistinguishable from the lymph-corpuscles which are found in an inflamed cornea, and whose source it can be demonstrated is the extravasation from the conjunctival blood-vessels. I shall not recapitulate the observations and inferences on this subject which I have recently published at some length in a series of papers on inflammation in the ' Edinburgh Medical Journal,' but will state in a few words the kind of evidence which is obtainable on this point.

Solution of osmic acid fixes and stains colourless blood-corpuscles within and without the blood-vessels in such a characteristic manner that they can be recognised with certainty. If an inflamed frog's tongue, in which extravasation has been observed under the microscope, is treated by osmic acid solution and examined, the arrangement of the colourless corpuscles in some of the sections can be recognised as similar to that which was observed in the transparent living tissue. The identity is beyond dispute. Estimated by this criterion, the cells which fill the spaces which are adjacent to the epithelial growth in a cancerous skin are extravasated colourless blood-corpuscles. When it happens that the section includes a blood-vessel, and a colourless corpuscle can be seen amongst the red corpuscles within the vessels, the absolute similarity between the colourless blood-cell within the vessel and the cells between the bundles is unmistakable. No stronger proof can be given

unless the passage of the same cell could be followed from the vessel to the interstices of the tissue, which in the circumstances is manifestly impossible. To any one who will study the appearances of colourless blood-corpuscles when acted on by osmic acid this direct proof is as unnecessary as it is impossible. There is no other cell product with which I am familiar, certainly none connected with the phenomena of inflammation, which can be confounded with colourless blood-corpuscles in an osmic acid preparation.

Between the small rounded cells at the outskirts of the infiltration and the cells immediately adjoining the growing epithelium there is a difference in size and a difference as regards the more complete development of the nucleus. As regards both differences the transitions are very gradual. The nearer the epithelium, as a general rule, the larger the cell and the more completely developed the nucleus, but amongst the more developed cells are nearly always to be found some of the smaller cells which have undergone no change since they were extravasated from the vessels.

The development of a well-formed rounded nucleus is not dependent on the increased size of the cell, as in the small round cells amongst the bundles a greater proportion contain a round, single, fully formed nucleus than is the case with the lymph-cells in simply inflamed tissues. And here I would remark parenthetically that the hypothesis that the lymph-cells with multiple small nuclei are doomed to destruction is unproved. Observations of my own, which are still unpublished, lead me to believe that the multiple small nuclei which are found in many lymph-cells combine to form the single large nucleus which is found in other lymph-cells. According to this idea, the large number of lymph-corpuscles in cancerous infiltration which contain a single well-formed nucleus is due to a characteristic development of the cell after it has left the vessels.

The changes of which the lymph-corpuscles effused in a

cancerous tissue are susceptible, and which many of them undergo, are an increase in the size of the cell and a development of the nucleus. That the latter is not dependent on the former is shown by the fact that an unusually large number of the corpuscles have a round complete nucleus before they have increased at all in size. In some instances, indeed, the nucleus constitutes nearly the whole of the cellular body.

The relation which these cells bear to the epithelium of the cancerous growth is an intimate one, and forms the foundation of the explanation I offer regarding that part of the morbid process which seems to me to constitute to a large extent its essential nature, namely, the continued extension of the epithelium by the formation of new epithelial cells.

The colourless blood-cells are in contact with the fully developed epithelium in three ways :

First, the contour of the epithelial growth may be sharply defined, an appearance indicating a delicate membrane forming a border or hem separating it from the contiguous elements. Adjacent to the epithelium one or two rows or layers of colourless cells are seen, which combine the appearance of epithelial with that of lymph-cells. They are continued indefinitely into the typical lymph-cells which fill the interstices of the tissue.

Secondly, the epithelial cells may be seen continued gradually into the lymph-cells, it being impossible to say where the one kind of cell ends and the other begins. The epithelial cells get smaller as they are nearer the edge of the growth, the outer parts of which are constituted by simple lymph-cells. In some preparations it is possible to observe small spindle cells among the lymph-cells, and they can be traced into the fully developed epithelium.

Thirdly, in describing the changes that the epithelial cells of the cancerous growth undergo I mentioned that some of them degenerate until either the nucleus is repre-

sented by a vacuole, or both nucleus and cell-substance may have disappeared, and nothing of the original cell is left but two concentric horny rings corresponding to the contours of the nucleus and the cell. Or all trace of the contour of the nucleus may have disappeared, and one such ring may be left corresponding to the contour of the cell. Into the vacant spaces thus left one or more lymph-corpuscles may enter, and fixing themselves in the position of the dead cell may develop into one or more epithelial cells which fill up the gap left by the withering of the previous occupant of the space.

The lymph-cells amongst the epithelium which are still unaltered can be identified by their size, the manner in which they are stained by carmine and logwood, and by the effect on them of osmic acid, in all of which respects they differ from the epithelial cells by which they are surrounded, and agree with the lymph-cells which are present in the spaces of the fibrillary tissue, and which are clustered round the extending epithelium.

Amongst the newly formed epithelial cells which have reached the full epithelial development transition cells from lymph-corpuscles to epithelium are not usually found deeper than in the two last-formed layers.

The thin membranous band which I have described as bounding the epithelial growth may be sometimes seen partially detached, and a connected mass of lymph-cells can be traced from the subepithelial tissue through the gap and into the fully developed epithelium.

Those lymph-corpuscles which are in the immediate neighbourhood of the epithelium partake to a certain extent of its characters, but when they are brought into direct contact with it they acquire all the characters of a fully developed epithelial cell.

The mode of development of the epithelial cells in cancerous tissue is intimately connected with that of the physiological regeneration of epithelium in healthy tissue and after destructive inflammatory processes. I have

made a series of investigations into the nature of the regenerative process in both these conditions.

In confirmation of what I have stated regarding the epithelial growth in cancer I cannot do more here than remark that the results of my researches, in so far as they have yet extended, accord with the opinions of those histologists who believe that the regeneration of epithelium takes place invariably by a special development of colourless blood-corpuscles.

The general conclusions at which I have arrived may be shortly expressed as follows :

In this disease there is an abnormal growth of epithelium and a morbid condition of the fibrillary tissue of the cutis.   I do not know which of these factors is the earlier in point of time or the most important in point of development, but I am of opinion that the change in the fibrillary tissue is not due to pressure by the encroaching epithelium.

When the blood-vessels of a papilla are choked by the growing epithelium the destruction of the fibrillary tissue in the papilla is probably hastened, but in the cutis, long before any pressure can be exercised either on the vessels or tissue, the bundles have begun to undergo absorption. There is evidently a morbid influence in action here, which is totally distinct from the mere growth of the epithelial cells.   Whether they both spring from a common cause or whether the one is the cause of the other is not apparent.

The extravasation of lymph-corpuscles into the spaces of the tissue is suggestive of the similar phenomenon in inflammation, and the mechanism by which it is called into action and effected is probably the same.   It is quite conceivable that the abnormal epithelium may act as a foreign body, and the effects of the injury it inflicts being propagated to the vessels, the weakened walls permit the extravasation.   But there is an important difference in the cancerous as compared with the inflammatory change. Not only are the ordinary clinical symptoms of inflamma-

tion absent, but its products do not follow. The material poured into the tissue from the blood does not organize. No newly formed connective tissue takes the place of the absorbed and degenerating bundles, a fact which has been already commented on by an author whose name I cannot at present recall. The plasma is either deficient in quality or its power to form the tissue is destroyed by some morbid agent developed in the part.

I am not inclined to attribute with the author to whom I have alluded this defective quality to a condition that affects the whole blood, because it is a matter of everyday experience that when a cancerous tumour is removed, in the wound so produced the healing process may be rapid and satisfactory. I am forced, therefore, to suppose that there is something in the cancerous part which is prejudicial to the formation of new tissue, and whatever that may be it is very probably to the same cause that the degeneration of the previously existing fibrillary tissue is to be attributed. This unknown agent may be a product of the abnormal epithelium, or both the abnormal epithelium and the degeneration of the connective tissue may, as I have already suggested, be due to a common cause.

The development of lymph-corpuscles into epithelium, when brought into contact with the epithelial cells already existing, being a process that, I believe, takes place also in healthy tissue, cannot be considered by me as in itself peculiar to cancer, but the abnormal extent to which the process develops is characteristic of the disease. This is shown, not only by the increased development which takes place in the immediate neighbourhood of the epithelium, but by changes in lymph-cells at a considerable distance from it, which are of a more or less marked epithelial character. The changed character of the large oval nuclei which I have described as being seen in the corium may be, perhaps, attributed to a similar influence.

We can only judge of the nature of this influence by its effects, and these we find to be analogous in one important feature to a change which is constantly taking place in

physiological conditions.　In health I believe that a lymph-corpuscle which is in contact with epithelial cells becomes assimilated to the cells with which it is in contact, and that this is the source of the regeneration of the epithelium, the area in which this change can take place being strictly limited.　In cancer, on the other hand, the potential epithelial area extends along the lymph spaces of the adjacent tissue, and the lymph-cells that are present in it are subjected to a developmental change that they do not undergo in health except when in direct contact with normal epithelial structures.　This influence, or epithelial infection as it has been termed, may exist both in the fluid and formed elements of the affected tissue, but that it is strongly present in the cells may be inferred from the rapid epithelial transformation which rows of cells undergo when they form an unbroken chain which is at one end in contact with the epithelial growth.

How much of the intensity of this influence is due to a more powerful action of the substance, whatever it may be, that produces epithelial growth, and how much to a diminished power of resistance on the part of the lymph-cells, it is impossible to say, but both conditions are probably present.

The absorption of the fibrillary tissue which I have described as a change distinct from the growth of epithelium is probably produced by contact with a substance generated in the part, as it may be arrested for a period of years by a timely operation.　This substance can only act on the tissues of the individual in whom it has been generated.　The non-inoculability of cancer which may be inferred from the complete immunity from infection that is enjoyed by surgeons and nurses points to an abnormal condition of the tissue, which predisposes it to succumb to the morbid influence.

I have thus arrived at results similar to those obtained by Classen in the cancerous cornea, and as regards the growth of epithelium from colourless blood-cells in physiological conditions I am in accord with the views expressed

thirty years ago by Addison and more recently by Biesia-
decki and Pagenstecher.

As far as my investigations enable me to judge, the
views I hold regarding cancer of the skin are applicable
to cancer of other organs. With the exception of scirrhus
mammæ these have, however, been limited, and in the case
of cancerous breast I rest my opinion more on the instruc-
tive nature of osmic acid preparations than on the number
of tumours examined.

Since the foregoing remarks were written a paper has
appeared in ' Virchow's Archiv ' (66 Band, 2e Heft) on
the development of cancer in the diaphragm as a secon-
dary growth, by Arcadius Rajewsky, and as the author
states that his views regarding the nature of the process
in the diaphragm are applicable to cancer of the skin,
which he had also investigated, although details of the
investigation are not given, it will not be out of place if
I add a few words regarding his memoir. His observa-
tions were made in Von Recklinghausen's laboratory, and
it is to be presumed that the views expressed represent
those of that pathologist.

He finds that cancerous epithelium extends from the
lymphatic vessels to the *Saft Kanälchen,* and that it is
first formed by a proliferation of the lining epithelium of
the vessel. The newly formed cells proliferate onwards
and onwards in the lymph-channels. The substance of
the bundles of tissue undergoes absorption. This observer
is thus at one with myself regarding the relation which
the newly formed cells have to the bundles, as the *Saft
Kanälchen* are equivalent to the spaces which I have
described as existing between them. He further confirms
what I have stated in regard to the absorption of the
tissue as an independent part of the cancerous process.
He differs from me in believing that the growth of
epithelium takes place first from the lymphatic vessels.
My preparations teach me, on the contrary, that the new

epithelium may form wherever there is an epithelial surface pre-existing.

I cannot find that the proliferation of the lymphatic epithelium is proved by Rajewsky. In the drawing (fig. 2 in his plate) I can only see a number of cells in an intermediate stage towards fully developed epithelium, and I see no evidence that a cell is dividing into two or more cells, or that any one of the cells figured has been produced by any one of the others. The resemblance of the smaller cells between the bundles to lymph- or colourless blood-corpuscles had not escaped him. He remarks that the cells which are more distant from the cancer-centres might be taken for " wandering cells," that is, lymph-cells. He adds that such an admission as to their nature can hardly be made (although he does not explain why), and considers it more probable that they are products of such proliferation as is supposed to be represented in the drawing to which I have referred, and which I cannot see represents any such process as is indicated by that term.

I fail to see that this latest author has added anything that makes the proliferation theory less of an hypothesis and more of a fact than it has always been, while it is satisfactory to be able to claim such an authority as that of Von Recklinghausen in testimony of the impossibility of distinguishing between colourless blood-corpuscles and the first appearance of the cells that will afterwards become unmistakable cancer-epithelium.

In conclusion I have to express my thanks to Mr. Bell, of the Edinburgh Royal Infirmary, for the kind manner in which he has provided me with material for this investigation, and to Mr. Ewart for executing the accompanying drawings.

DESCRIPTION OF PLATE X.

All the figures are drawn from osmic acid preparations. The magnifying power is that of the No. 7 objective of Hartnack, with No. 3 eyepiece, which is equal to about 300 diameters.

FIG. 1.—The substance of the bundles of the fibrillary tissue has been absorbed, but the boundaries of the bundles (a) persist. The arrangement of lymph-cells (b) is still determined by that of the bundles.

FIG. 2.—A secondary bundle on which the nuclei of flat cells of the normal tissue (b) still persist. Lymph-cells (a) still retaining their characteristic appearance are seen on the surface of the bundle.

FIG. 3.—Primary bundles (b) which still present the normal appearance. Lymph-cells (a) between the bundles. Nuclei of the normal tissue (c) seen on the bundles.

FIG. 4.—Part of a section in the vicinity of abundant cell infiltration. (a) Cells of the rete Malpighii; (b) transparent membranous tissue adherent to the rete; (c) large oval nuclei of the normal tissue persisting; (d) a blood-vessel; (e) lymph-corpuscle (colourless blood-corpuscle) within the vessel.

FIG. 5.—Growth of new epithelium by apposition of lymph-corpuscles, between which minute spindle elements extend into the rete. (a) Cells of epidermis (prickle cells) in the horny condition; (b) cells of rete Malpighi presenting a normal appearance; (c) lymph-corpuscles; (d) spindle-cells.

FIG. 6.—Section of rete and subjacent tissue, torn in order to facilitate the examination of the individual cells. (a) Cells of rete Malpighii; (b) lymph-corpuscles in transition to epithelium; (c) lymph-corpuscles showing first stage of the epithelial change; (d) fibrillary tissue; (e) lymph-corpuscle unaltered.

FIG. 7.—Cells which have undergone horny degeneration. (a) Transparent homogeneous substance within a horny ring; (b) horny ring from which the cell-substance has fallen out.

FIG. 8.—Shows an early stage in the formation of a laminated capsule. (a) A swollen vitreous cell; (b) indicates a capsule of withered horny epithelium.

FIG. 9.—A cell in a sebaceous gland undergoing horny degeneration. (a) Horny ring round the nucleus, which is represented by granular débris; (b) edge of withered cell-substance which is separated from the nuclear horny ring; (c) horny ring surrounding the cell.

Plate X

FIG 3

F G 2

FIG 1

a

b

c

c

b

a

F.G 5

F G 6

b

e

FIG 8

FIG 7

FIG 9

a

b

a

a
b

b

a
b
c
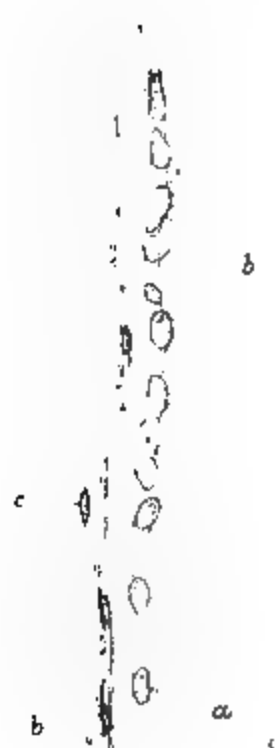
ON THE

DEVELOPMENT OF SPINDLE-CELLS IN "NESTED SARCOMAS."

BY

WILLIAM R. GOWERS, M.D.,

ASSISTANT PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND TO THE
NATIONAL HOSPITAL FOR EPILEPSY AND PARALYSIS.

THE object of the following paper is to describe the development of spindle-cells in certain sarcomatous tumours, and to direct attention to a structural peculiarity possessed by those sarcomas in which this mode of development was best observed.

The tumours alluded to were all, in their histological characters, good examples of spindle-celled sarcoma, and they presented that partial arrangement of the cells in tracts and bands traversing the growth which induced Virchow to term such forms "fasciculated." They presented also a concentric arrangement of the cells in nests, which bore a striking resemblance to the nests of epithelium-cells in epithelioma. Similar structures have been described by Sir James Paget[1] in a myeloid tumour of the skull.

[1] 'Lect. on Surgical Pathology,' p. 554. Third Edition.

Lebert[1] also described and figured a similar concentric arrangement of fusiform cells around the "mother-cells" of a myeloid tumour. These nests lay for the most part among the less regularly arranged cells between the fasciculi, but also in part within the looser fasciculi. In a scraping of a recent tumour they constituted a very conspicuous feature.

The spindle-cells of which these tumours were composed were of the usual form and size (Pl. XI, figs. 3, 4, and 5). They varied in length from $\frac{1}{1000}$ to $\frac{1}{300}$ of an inch, and in width from the $\frac{1}{4000}$ to be $\frac{1}{3000}$ of an inch, and contained, in their widest portion, an oval nucleus having an average long diameter of $\frac{1}{3500}$ of an inch. The nuclei were larger, in proportion to the size of the cell, in the softer and more rapidly growing parts of the tumours. In some of the older and harder portions the nuclei were hardly recognisable, and the cells were so linear as to resemble fibres until close examination showed their nature. In the softer portions the nucleus appeared to distend the cell, which tapered suddenly from each end of the nucleus.

These last-named cells were delicate in outline, and contained finely granular contents. The nuclei contained many coarser granules, one or two of which were very large. No more distinct nucleolus could be recognised. In fragments of the softer portions of the tumours many similar nuclei were seen free in the field of the microscope, sometimes surrounded by a little granular protoplasm. In these parts there were in addition globular cells about the $\frac{1}{1000}$ inch in diameter, possessing similar nuclei. Cells could also be seen in two of the tumours in process of development from the globular into the spindle shape, by the process to be immediately described, and in the softest tumour examined these intermediate forms were exceedingly abundant.

The *nests* of cells were composed of such spindle-cells arranged concentrically (Pl. XI, figs. 3, 4, and 5). They were globular or slightly oval in shape, and varied in size

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[1] 'Path. Anat.,' pl. xxvii, fig. 5.

from the $\frac{1}{1000}$ to the $\frac{1}{200}$ of an inch. In some the concentric arrangement obtained almost up to the centre of the globe ; in others the centre was occupied by nuclei embedded in granular protoplasm, and resembing the "mother-cells" of myeloid. The outer portion was studded with nuclei similar to those in the centre, and among the nuclei concentric lines extended, clearly due to the outlines of the fusiform cells to which the nuclei belonged. On the outer surface the most external cell was in many cases partially detached. The smaller nests consisted of three or four cells only, the outlines of which were very distinct. In some instances two series of concentric cells were enclosed in a similar common capsule (fig. 2 f)

In three tumours possessing such a structure the growth was intra-cranial in each case, and sprang from the inner surface of the dura mater. In each case the tumour was globular in form, and had attained the size of a small orange. The surface was nodular. Each tumour had displaced, but had not invaded, the brain substance. The convolutions were pushed away on each side and narrowed by pressure, and the tumour lay in a hollow in the brain which it had formed by its growth ; on the surface of the hollow the grey substance had disappeared, being atrophied from pressure, but the growth and the cerebral substance were nowhere continuous. The consistence of the tumours varied, the older portions, near the origin at the dura mater, were very firm, but the more recent parts were much softer. In one specimen the whole tumour was soft, not firmer than brain tissue. Each specimen was greyish in colour, the grey being abundantly mottled with•red from distended vessels and extravasations in the softer portions. The soft tumour just mentioned resembled in tint and consistence grey cerebral substance. The section yielded abundant " juice," containing free cells and nest-masses.

It was in the last-named growth that the origin of the spindle-cells, of which it was mainly composed, could be traced with great distinctness, but in one other of these

tumours the same origin of the cells was distinct. The process consists in the development of spindle-cells from small spherical cells by the process of so-called endogenous cell development, which has been described by Dr. Creighton[1] under the name of "vacuolation." It was long ago pointed out by Sir James Paget[2] as occurring in epithelial cells, but has, till lately, received little notice.

The stages of the process are inferred from a comparison of the appearances presented by cells in what appear to be different stages of development. Each cell-form shown in fig. 1, Pl. XI, could have been multiplied many times from every specimen placed under the microscope.

The starting-point of the development is a small round cell, variable in size, but about the $\frac{1}{1500}$ inch in diameter, with a delicate cell-wall, finely granular cell-contents, and a round, or more commonly oval nucleus, about the $\frac{1}{2500}$ inch in its longer diameter, cell and nucleus having the characters already described. At first, the nucleus is often in or near the middle of the cell (Pl. XI, fig. 1, a), but if not at first it soon becomes excentric, and in the part of the cell away from the nucleus a clear space appears in the midst of the granular protoplasm (fig. 1, b). This space, which is circular, is quite clear. It increases in size, and as it increases in size the granular cell-contents, at first occupying the whole cell, become confined to its periphery, and are always most abundant on the side on which the nucleus is placed. The nucleus now lies quite against the wall of the cell. The granular protoplasm sometimes forms a ring around the vacuole (fig. 1, c*), sometimes lies upon it as a crescent, the clear space touching the cell-wall on the side furthest from the nucleus (fig. 1, c). The granular protoplasm then tapers off to a point on each side of the nucleus, the points being connected by the boundary wall of the original cell. At this stage the cell-form has been compared very aptly by Rindfleisch to a signet-ring. It is, in fact, a spindle cell

[1] 'Reports of the Medical Officer of the Privy Council,' 1874 and 1875.
[2] Loc. cit., p. 719.

lying around a clear central space. The inner side of the spindle cell thus formed has not at first a sharp limiting line, but this it acquires. The cell then peels off the central clear body, first one point becoming detached (fig. 1, e), then half the cell, then the whole. During this process the outline of the central clear mass is preserved; it is bounded by a delicate sharp line, and the substance is clearly different in tint from the fluid in which it lies (in these observations it was a ·75 per cent. solution of chloride of sodium). Several cells were seen in which the process of separation was incomplete, one half of the spindle-cell having become detached, the other half still being curved round the hyaline sphere (fig. 1, f).

In one cell figured the annular protoplasm had become broken and partly detached, while the hyaline "vacuole" was still in appearance central (fig. 1, h). It is to be noted that, even at this stage, the hyaline sphere has a distinct defined outline, while the protoplasm detached from it has none upon its inner side, that which had been applied to the hyaline sphere. This is, I think, a point of interest in reference to the nature of the process. In most of the free cells, which were formed and separate, a curve persisted, corresponding to that impressed upon them in their development, and it was to be noted that their boundary wall on their concave side was much less sharply defined than on their convex side, the two characters together showing that they had all arisen in this manner. The curve which this mode of development confers on cells is very frequent in sarcomatous and other tumours. The process, as seen in its simpler form, is thus one rather of cell-transformation than of cell-multiplication.

The relation of this process of cell-development to the formation of the concentric nests could be traced, in the same tumour, with great distinctness. The concentric arrangement of the cells appears to depend on the vital energy of the nucleus of the original cell, determining its early and repeated multiplication. Where the nucleus

remains single and undivided, the single spherical cell is developed into a single uninucleated fusiform cell. If the nucleus divides after it has acquired its lateral position, and is excluded from the portion of cell occupied by the vacuole (fig. 1, *k*), the two nuclei remain in contact in the resulting spindle-cell, which differs from others only in containing two nuclei instead of one (fig. 1, *l*). But the nucleus may divide before or very soon after the appearance of the hyaline area (fig. 2, *a*). In this case one of the nuclei only is included in the circumferential spindle-cell, the other remains, surrounded by some of the granular material, within the hyaline area. Several cells are sketched in this stage of development (fig. 2, *b*). All forms suggested that the nucleus and protoplasm around it may escape from the vacuole and from the embrace of the circumferential cell, and form a spherical cell in which a fresh process of vacuolation may develop another spindle-cell, and that this escape occurs where the spindle-cell already formed occupies only a part, and not the whole, of the circumference of the original cell. In other cases, however, the second nucleus remains within the cavity, and, with its protoplasm, becomes applied to the inner surface of the circumferential cell (fig. 2, *b*), within which it develops into a similar spindle-cell. Its nucleus is sometimes placed opposite that of the first cell, more frequently at some other part of it, so that the two cells, overlapping, complete a circle. I was not able to trace distinctly the relation of this second cell to the vacuolar process. The cell appeared to arise within the hyaline space, without any secondary vacuolation of its own granular protoplasm, but this might have occurred and easily have escaped recognition ; no cell forms were seen in that stage. In many cases, before the second nucleus thus developed, it again divided (fig. 2, *b**), one of the resulting nuclei developed into the inner cell, the other remained and multiplied within that cell (fig. 2, *c* and *d*). This process, occurring repeatedly, resulted in the formation of a number of cells, arranged

concentrically one within the other around a central mass of granular protoplasm, containing one or, more commonly, many nuclei. In some examples the persistence of the vacuolation was very distinct, a clear area occupying part of the space enclosed by the concentric cells, the rest of the space being occupied by granular protoplasm containing nuclei (fig. 2, *e*). In this way all these nests of cells appeared to have been developed. It is easy to understand how such forms arise as that in which two small nests were contained within one larger shell of cells (fig. 2, *f*), by two independent centres of vacuolation in the protoplasmic contents of a nest in the early stage of its development. In one of the oldest tumours examined a nest was seen in which the centre had apparently undergone calcification (fig. 4, *a*).

This process of vacuolation certainly plays a very important part in the transformation of the tissue elements of many morbid growths. It is only seen distinctly in the rapidly growing portions, and it is worthy of note that, at any rate in the tumours I have been describing, it could only be recognised in the fresh specimen. Commencing decomposition and the action of so delicate a preservative agent as bichromate of potash alike obscured the process. After the tumour had been kept for a few days in a 2 per cent. solution of bichromate of potash the distinctive features of the vacuolation were irrecognisable. In those cells which were in the early stage of the process the whole of the hyaline area appeared to have become granular; in those cells in which the process was nearly complete the preservative agent appeared to have caused the separation of the spindle-cell and the destruction or disappearance of the hyaline " vacuole," or else had caused the latter to shrink into very small dimensions as an irregular faintly granular mass attached to the concave side of the spindle-cell.

In the allusions to the process which occur in works in pathological histology it is commonly described as a vesicular change in the nucleus of the cell. I could find,

however, no confirmation for this opinion in the cell-forms observed. In each case the vacuole appeared to arise in the granular protoplasmic cell contents away from the nucleus, and in the earliest stage its limit was not sharp but gradual. This also appears to be the opinion of Dr. Creighton.

What, then, is the nature of the process? Is its essential element the development, within the granular protoplasm, of a hyaline body or vesicle which merely pushes the granular protoplasm before it on all sides? Or is to be regarded as a primary movement of the organic granules, with the nucleus, to the periphery of the cell, the clear area being merely that which is left by their departure? Each view would find some support in the facts of the process as just detailed.

Whether the " vacuole " be in the later stages a mere fluid-containing vesicle or not, it appears to consist in its early stage of transparent protoplasm, capable of becoming granular by decomposition. It is significant also that when a second nucleus has developed (by division of the first), this second nucleus with some attached granular protoplasm may be included within the vacuole. But the extreme tenuity which the circumferential spindle-cell ring may attain renders it difficult to resist the impression that there is an active distending force concerned in the process. It is also noticeable that the hyaline " vacuole " has a distinct sharp boundary line or wall, preserving its form under exposure, at a stage when the inner aspect of the granular protoplasm, which surrounds it, is on its part devoid of any such limitation. This is clearly indicated in one of the figures sketched, in which an imperfectly developed cell has been separated prematurely.

Cell-forms obviously due to vacuolation have been figured by many writers, but certainly often without due appreciation of the relations of the process. They show, however, that it plays an important part in histological changes of many kinds and in many tissues. I may

refer, for instance, to the figures of "bladder-bearing" cancer-cells ("Physaliphoren") by Virchow; of colloid degeneration of cells by Rindfleisch, and by Cornil and Ranvier; to the formation of pus-cells within vacuolated epithelium-cells by Rindfleisch; and to the distension of vacuolated cells with fat which is figured as occurring in the liver by Rindfleisch, and in inflamed medulla by Cornil and Ranvier. The laborious researches on the transformation of liver and epithelial cells by Creighton have been already alluded to.

Whatever be the real nature of the process, it is clear that vacuolation, in many cases at least, terminates the active life of the original cell. If the nucleus has not multiplied before or soon after the commencement of vacuolation it remains single, or its division is ineffective in cell-multiplication. The process thus accompanies or succeeds, it does not precede cell-multiplication, and its chief effect, in these tumours, seems to be to convert a growing round-cell into an inert fibre-cell, to transform the active into passive tissue-elements.

DESCRIPTION OF PLATE XI.

Development of spindle-cells, by W. R. Gowers, M.D.

FIG. 1.—The development of spindle-cells from round-cells by vacuolation : (from large soft sarcoma). *a.* Round nucleated cells before vacuolation has commenced; *b,* early stage of vacuolation; *c,* further stage; the original cell nucleus and substance a spindle-cell curved round the " vacuole"; *d,* the extremities of the spindle-cell separated by the vacuole; *e,* commencing separation of spindle-cell, which in *f* is nearly accomplished, the " vacuole" persisting; *g*,* separated cells preserving their developmental curve; *h,* cell ruptured before the end of the process; *j,* cell separated before the development of an inner wall; *k,* nuclear multiplication after vacuolation resulting in a binucleated spindle-cell as *l.*

FIG. 2.—The development of nests of spindle-cells (from large soft sarcoma). *a.* Multiplication of nucleus before commencement of vacuolation; *b,* inclusion of nucleus with granular protoplasm within the vacuole; *b*,* division of included nucleus within slightly granular vacuole; *c,* development of second spindle-cell within the first, a third nucleus remaining; *c*,* multiplication of such a third nucleus; *d,* a resulting nest with vacuole persisting; *e,* ditto of larger size with many nuclei in protoplasmic centre; *f,* two centres of cell development within a concentric series of cells.

FIGS. 3 and 4.—Cell forms and nests from a large spongy sarcoma. *a,* Nest, the centre of which has undergone calcification. *b.* Cells separating from exterior of a nest.

FIG. 5.—Cells from similar sarcoma. *a.* Cell developing by vacuolation.

Plate XI.   Med Chir Trans Vol LIX

FIG 1

FIG 2

FIG 3

FIG 4

FIG 5
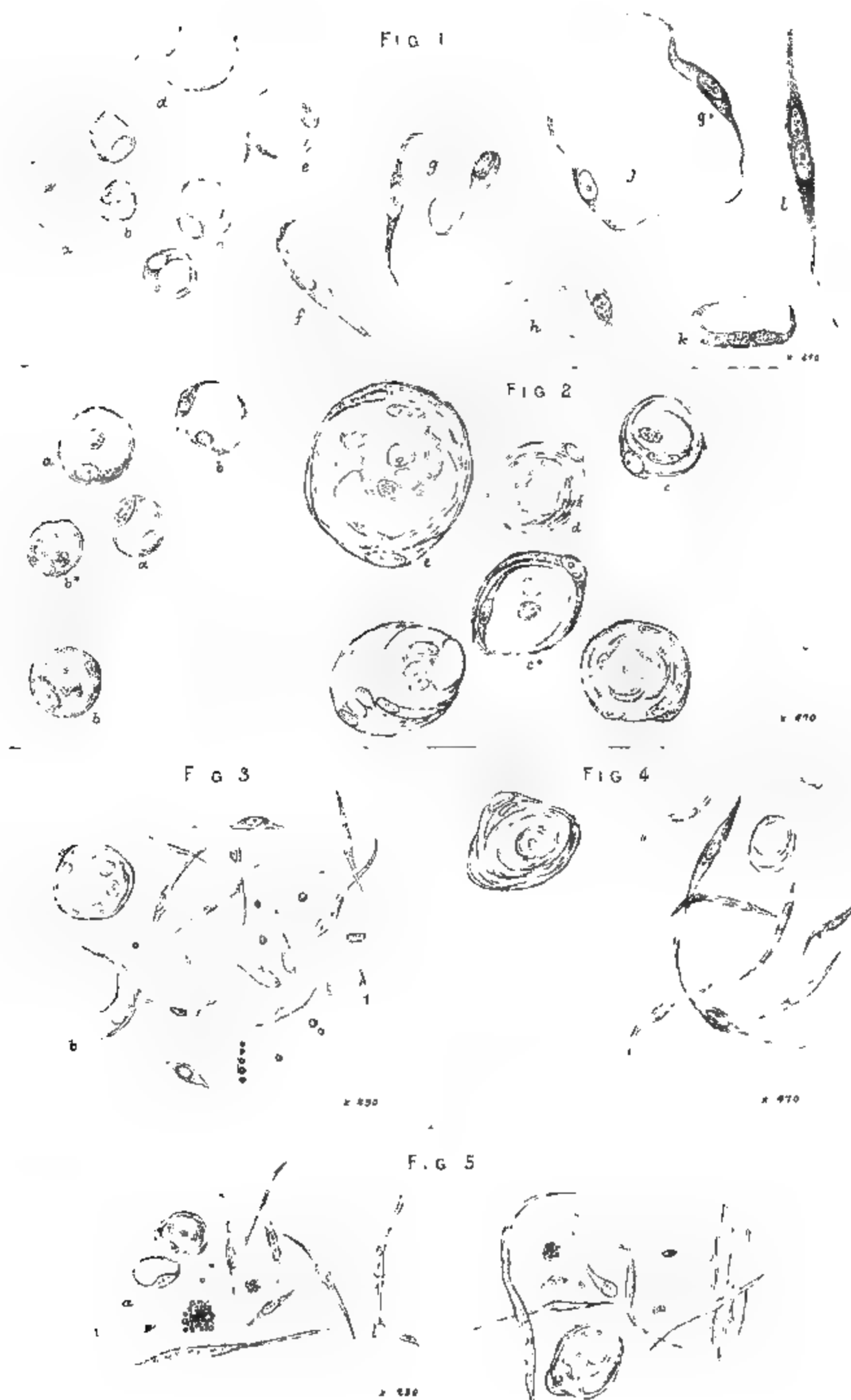
ON A CASE

OF

INTERMITTING HYDRONEPHROSIS;

WITH

SOME REMARKS ON HYDRONEPHROSIS AS A CAUSE OF ABDOMINAL TUMOURS.

BY

HENRY MORRIS, M.A., M.B. LOND., F.R.C.S.,

SENIOR ASSISTANT SURGEON TO, AND LECTURER ON ANATOMY AT, THE MIDDLESEX HOSPITAL.

IT is long since a special name was used to signify distension of the pelvis of the kidney, or pelvis and ureter, by retained urine. Martineau in 1785 called it " Hydrops Renis," and James Johnson in 1816 suggested " Hydro-renal-distension." In a recent volume (twenty-third) of the Pathological Society's ' Transactions ' the indefinite name " cystic tumour of the kidney " is used and defended; but since the publication of the ' Traité des Maladies des Reins ' in 1841, the term " Hydrone-phrosis" then introduced by Rayer has been very generally employed.

A word of explanation is, however, requisite concerning the prefix I have ventured to apply to Rayer's term in the heading of this paper, because the name " inter-mitting hydronephrosis " is not to be found in any

medical writings, so far as I am aware, except in a recent communication to the 'British Medical Journal' by Dr. Cole, of Bath. By it I mean a tumour caused by dilatation of the pelvis of the kidney, and it may be of more or less of the ureter, which disappears altogether for a time, to appear again and again at intervals. It is used only in a clinical sense, and even thus is not meant to be applied to cases in which the tumour varies only slightly from time to time—varies, that is to say, to such a degree only as may be due to the changes in the abdominal contents by the ingestion of food or irregularity of the bowels.

Pathologically, no doubt, the amount of distension in most cases of hydronephrosis is subject to variations; and in some, probably, such as where the dilatation is the result of pregnancy or carcinoma uteri, the pelvis of the kidney occasionally quite empties itself; but it would be incorrect to speak of any tumour or swelling which is not capable of clinical examination as *intermitting*, since any changes in size could not possibly be detected with certainty, and could only be inferred with hesitation.

The following case came under my care in the Middlesex Hospital in the autumn of 1874:

Jane G—, of Henry Street, Hampstead Road, æt. 56, a married woman who had had no children, was admitted into Regent Ward, on August 5th, 1874.

History.—Six years ago she began to suffer pain in the abdomen, especially about the bladder, and to pass blood in the urine which she voided with great difficulty. She was very ill for a time, and was told by her doctor that she suffered from inflammation of the bladder. She never thoroughly recovered, and up to two years ago

In March she applied for relief at the out-patient department of the Samaritan Free Hospital, but attended only for a very short time. After an interval of four months she returned to that institution on July 2nd, and was treated for cystitis up to the 23rd of the same month. At both these periods the symptom she mostly complained of was inability to hold her urine.

On admission into the Middlesex Hospital it was noted that she was a thin, bony-framed woman, with a pinched and distressed look about her face. She complained of pain of a "bearing-down" description in the urethra and vagina after micturition, but had no difficulty in passing urine. She stated that blood came away in her water, but thought that more flowed after than with it.

Stone or some vesical growth was suspected, and it was accordingly proposed to sound her, but on examining *per vaginam* a small, soft, vascular, papillary tumour about the size of a pea was seen at the orifice of the urethra. It seemed to be attached to the greater part of the circumference of the canal. The uterus and vagina were quite healthy. Blood, pus, and vesical epithelium, but no crystals, and nothing of the nature of a new growth, were present in the urine.

On August 8th a saturated solution of chromic acid was applied to the little tumour, and for the next day or two she passed less blood, and expressed herself as feeling much better, as she had no pain on micturition.

On August 11th the chromic acid was again applied, after which she was decidedly better, suffering no pain and passing but little blood. From August 15th to the second week in September my colleague, Mr. Andrew Clark, watched the case for me.

On August 26th the notes ran—"Not so much blood in urine; has some pain on micturating; otherwise better."

On September 8th a large quantity of blood was passed in the urine and she complained of severe pain in the back and abdomen. The bladder was examined and a suspicion of some growth in it was strengthened by a

certain unevenness of the surface being felt by means of the sound.

On September 19th I made a vaginal examination and found a vascular tumour the size of a large hazel nut projecting from the meatus urinarius. It must have presented itself here quite recently, as no trace of it was seen at the preceding examination. It was very sensitive to touch, which, in fact, gave rise to very acute pain. It was pedunculated, and so far as one could judge was attached near the neck of the bladder, just above the urethral orifice. The urine at this time was acid, contained about one fifth of albumen, but no blood; severe pain was complained of in the back and about the hypogastrium.

On September 29th chloroform was administered, and the tumour of the bladder was removed with a fine wire écraseur. The urethra was so dilated that the little finger was passed without difficulty into the bladder, but the point of attachment of the growth could not be felt. Soon after the operation she lost all pain on micturating, but the urine was constantly running from her. She retched a good deal and could keep nothing but ice on her stomach. The urine was albuminous but not tinged with blood.

On October 3rd sickness continued; there was no pain about the pelvis, but much was complained of in the *right* hypochondriac and lumbar regions. On examining the abdomen a distinct rounded tumour was felt in these regions, about the size of the head of a small foetus. The patient was unaware of its existence. The outline was well marked except above, where it passed to the under surface of the liver, and even seemed to be connected with it. The urine contained no pus nor blood, and was passed painlessly though still involuntarily.

On October 4th, having asked Mr. De Morgan to examine the tumour in the loins, he was about to do so, but it was found to have quite disappeared.

On October 6th the tumour was again distinct. The

vomiting still continued, and the vomited matter was of a greenish character.

On October 9th the retching was very frequent, but the patient had been able to keep down one or two oysters, some chicken-broth, and milk. A simple enema was ordered, as her bowels were very constipated.

On October 10th the enema had had the desired effect, a large quantity of solid fæces had been passed, after which the tumour disappeared.

On October 11th the body, face, and arms were covered with a crimson exanthematous rash. Hiccough was troublesome. Another enema had been given, as she felt she required a further action of the bowels. She had been taking two oysters daily since the 8th; these were to be discontinued.

The next day the rash had almost disappeared, and the sickness and hiccough were better.

From this time to her death, on October 22nd, there was no important change to record. Sickness, a constant symptom, was sometimes more, at others less severe; the bowels were always constipated, and the fæces very hard; the urine was always passed involuntarily, and when once or twice a little was obtained and examined there was no blood in it. She was always restless at night and thirsty, but during the day dozed a good deal.

The abdominal tumour varied frequently; some days being distinct and even prominent, and in this state was more than once examined by Mr. De Morgan and others; then it altogether disappeared, to return again and be as evident as before. It did not give the impression of being firmly fixed, but it did not float, nor could it be moved appreciably from its position in the right lumbar region.

Autopsy.—An examination of the body was made twelve hours after death by Mr. Clark and myself. There was nothing requiring notice about the thoracic organs. The liver and spleen were healthy. The *right kidney* was much enlarged, and its pelvis greatly distended with urine, so that the whole formed a considerable tumour occupying

the right hypochondriac and lumbar regions. To the front surface of the distended pelvis, a portion of the small intestine was adherent by means of a strong broad band of old false membrane. The colon was not adherent to the cyst. The kidney, as well as being hypertrophied, was much congested; many of the tubes were surrounded with pus-cells, and near the surface were a few circular purulent depôts. The *left kidney* was very small, the pyramidal portion being entirely destroyed, and the cortical part much wasted, though unequally so in different parts; its pelvis and ureter were dilated, but less so than those of the right.

All the pelvic organs except the bladder were healthy. The *bladder* was contracted, and the trigone very small; there was no disease of the tissues between the orifices of the ureters and the urethra. Above the orifice of the right ureter was an oval-shaped, raised, but not pedunculated mass, warty-looking, and fissured upon the mucous surface, and of the same creamy colour as the growths on the left side; it extended upwards some distance on the right postero-lateral wall. The *right ureter* was pervious throughout; its vesical orifice was not, however, dilated.

The orifice of the *left ureter* was much dilated and admitted the uncut end of a cedar pencil; the mucous membrane of its margin was somewhat overhanging all around. Situated just above it was a pendulous vascular outgrowth three quarters of an inch in length, which hung over it like a valve; its free extremity was of a blackish charred appearance. To the median side of this pendulous mass and just above a line joining the orifices of the ureters was a nodule the size of a marble, with a broad base, projecting into the cavity of the bladder; it was firm in consistence, not vascular looking, but of a creamy white colour both on the surface and on section. Above and to the left of this for some distance the mucous membrane was infiltrated by new growth, which was raised and presented a fissured surface, the depressions or cracks of which had

also a moist cheesy or cream-coloured appearance. Attached near the summit of the bladder on the left postero-lateral aspect and hanging into the cavity by a long slender pedicle was another but smaller vascular outgrowth. The free end of this was torn and charred-looking, like that attached near the orifice of the left ureter.

The kidneys and bladder were examined microscopically by Dr. Coupland, who reported as follows:

Thin *sections of the large kidney* show large collections of pus-cells between and around the tubules obscuring the latter. Numerous fat-globules are also to be seen.

The *small kidney* shows atrophy of tubules with a considerable excess of interstitial fibrous tissue.

The *bladder* was covered on its mucous surface by villous outgrowth composed almost entirely of epithelium. The villi, though owing to their extreme brittleness they were broken short, showed evidence of being branched at their periphery. The submucous tissue was infiltrated with shrivelled and variously formed epithelial cells which occurred in groups between the bundles of connective tissue. In some places a small round-celled (granulation cells) infiltration replaced the epithelial growth. The characters ally the disease to papilloma or " villous cancer " of the bladder.

Remarks.—The actual nature of this intermitting tumour was not correctly diagnosed during life; but, it must be
t is pathognomonic of hydro-
any rate was not known to
discharge of a large quantity
idence of the tumour. The
umour alone is not sufficient
sis; it might have been an
or a cystic tumour of some
are known to undergo great
discharge through the Fal-
a common with other cystic
ntents into the bladder or

The diagnosis could have been made certain only by the discharge of urine in large quantity at the time of or soon after the disappearance of the tumour, or by the discharge of pus in the urine, in association with previous renal symptoms.   In the case of *Jane G*—, however, after the discovery of the tumour, and for some days before, the bladder could not retain urine so that no relation was made out between the quantity passed and the prominence of the abdominal tumour.   No difference in the quantity of urine was noticed from day to day by those whose duty it was to keep the patient dry.

Etiology.—By the light of the post-mortem examination it is not difficult to explain the origin of the tumour.   It is pretty certain that the villous disease of the bladder dated back to the patient's severe illness six years before admission into the hospital; how much longer it had existed cannot be stated, but we know that such growths are often of very long standing.   The left side of the bladder was the most diseased, and from it were suspended the two pedunculated masses.   These during the last few months of her life had given rise to much distress after micturition by being floated onwards with the urine to the urethral orifice, where they were gripped by the sphincter vesicæ. No doubt, too, her suffering was aggravated by the contraction of the bladder upon these highly sensitive structures; for that they were highly sensitive was proved by the pain excited by touching them, when first one and afterwards the other protruded through the urinary meatus. One of these masses, as I have said, overhung the orifice of the left ureter like a valve, and no doubt obstructed the flow of urine from the left kidney.

This obstruction slowly caused the dilatation of the left ureter and pelvis of left kidney, and by the constant compression it produced led to absorption of the secreting structure.   To compensate for the loss of function of the left kidney the right became hypertrophied.   Meanwhile the disease of the bladder increased and at last encroached on the tissue about the orifice of the right ureter and thus

obstructed the urine from the right kidney also. Although probably never quite complete on this side, the impediment was sufficient to distend the pelvis and ureter into a large cyst, and to set up interstitial suppurative inflammation of the kidney.

As the right kidney itself was much hypertrophied in addition to its pelvis being dilated it is easy to understand how a tumour was formed large enough to be detected through the abdominal parietes of a thin person; while the occasional subsidence of the tumour is to be explained by the forcing open of the compressed lower end of the ureter by the weight of the accumulated urine above.

There is no reason for thinking that the left kidney, although its pelvis was dilated, had ever given rise to a palpable tumour; nor is it indeed likely to have done so considering how atrophied it was, and how gradual must have been the obstruction.

We have here, then, a case of double hydronephosis, producing on one side an evident abdominal tumour, caused by the presence of villous disease of the bladder. It is the only case I am aware of in which the one condition must be considered the result of the other. Rayer in his very complete chapter on hydronephrosis gives an account of a man in whom these two conditions coexisted, but the hydronephrosis was produced by a calculus blocking up the vesical end of the left ureter. The left ureter, and the bladder near the orifice of the left ureter, were the seats of numerous small fungoid growth varying in size from a pin's head to a small pea. The calculus was formed of phosphate of lime coated with dried blood.

Some explanation seems to be required for the occurrence of an abdominal tumour in some cases of hydronephrosis and not in others. If we inquire into the causes of this morbid state we find that one of the most frequent is the pressure arising from cancer, and other diseases of the uterus, from enlargement of the prostate, and from stricture of the urethra; yet hydronephrosis, as a rule, is

only discoverable at the post-mortem examination, and it is a rare thing to find any sign of it during life.

Amongst the numerous cases of cancer of the pelvic organs treated annually in the Middlesex Hospital, none of the present surgeons[1] can remember to have seen a single instance in which distension of the pelvis of the kidney and ureter could be detected by the presence of an abdominal swelling during life; yet hydronephrosis is almost weekly seen in the post-mortem room of the hospital. Out of 52 cases of hydronephrosis collated by Dr. Roberts, of Manchester, six only were due to pelvic tumours (including cancer of the uterus), and he remarks, " Cases of this class are no doubt much more frequent than these numbers indicate; but they are generally slight in degree, and seldom go on to the production of a palpable tumour in the flank " (' Urinary and Renal Diseases,' p. 482). Howship, after discussing all the known causes of retention of urine in the ureters, agrees with M. Desault in his opinion that the most ordinary cause is scirrhous uterus, and he concludes by saying, " After all, it will generally happen that retention of urine in the ureters cannot be ascertained till after death, particularly if the affection be confined to one side, for in this case as the secretion declines in the one kidney, that of the other usually becomes more active and efficient " (' Practical Treatise on the Urine,' 1823, p. 214).

Rayer also considered (pp. 486-7, op. cit.) cancer of the uterus one of the most frequent causes of retention of urine in the ureters, and consequently of hydronephrosis and atrophy of the kidney; but he refers to three cases only which he had seen, and in only one of them could a tumour be detected. Even this was no exception to the rule, that cancer of the uterus does not lead to such distension as to give rise to an abdominal swelling, for in Rayer's case the tumour which occupied the right lumbar

* This was written before the death of Mr. De Morgan, late senior surgeon to the Hospital, whose experience of cancer was very unusually large. Mr. De Morgan's entire sanction was given to this statement as it here stands. He had read this paper before it was sent to the Society.

region during life was produced by compression of the right ureter by enlarged lymphatic glands.

I am thus led to the conclusion, from my experience of hydronephrosis in the dead subject, and after reading the description of about 50 cases of hydronephrotic tumours, that when an abdominal swelling exists there has been either sudden and complete obstruction to the urine secreted by a kidney in its full functions; or else that after one kidney has been damaged by disease, and the other has undergone compensatory hypertrophy, some obstacle has arisen to the flow of urine from the hypertrophied organ.

Instances of the first kind occur from the impaction of a calculus in the ureter, for example. Up to the time of obstruction the kidney had been rapidly secreting, it still goes on doing so, and the urine finding no exit dilates the pelvis and upper end of the ureter. Some idea of the force with which it does this can be obtained by remembering the conditions under which the urine is secreted in cases of complete retention, from stricture of the urethra; not only is the bladder distended to five or six times its normal size, but the viscera with their contents are greatly displaced, and a case has been recorded in which a portion of intestine was even expelled by the accumulating urine into the sac of a scrotal hernia. As the renal tissue becomes destroyed by compression the amount of fresh urine formed is lessened, but still the distension will increase, and the pelvis of the kidney will enlarge as long as there is any secreting structure left to add more urine to that already accumulated.

The case of Jane G— is an illustration of the second method; the right kidney was doing double work, so that when the obstruction arose the pelvis was distended with even greater force than that of a normal kidney would have been under similar obstruction, while the enlarged kidney assisted to make the swelling more prominent. Some further explanation must no doubt be sought for the enormous distension of the ureters, and the sacculation of the kidneys met with in such cases of diabetes insipidus

as are described in Beale's ' Archives,' by Dr. Strange, of Worcester, and Dr. Eade, of Norwich, but as in none of these a tumour was detected during life, I am not now concerned with them except to exclude them from the above remarks.

When, on the other hand, the obstacle is slight at first, and only gradually increases (as in cancer of the uterus and in organic stricture) there is a continual resistance rather than an actual obstruction to the flow of urine, so that the secreting structure becomes destroyed, *pari passu* with the distension of the pelvis of the kidney, and death occurs before the dilatation is sufficient to cause a tumour. In stricture, and in most cases of uterine disease, this resistance is opposed to both kidneys, and they each suffer, but cannot share, the results of the obstruction. Thus, though as Howship says there is less chance of detecting hydronephrosis in cancer of the uterus during life, " if the affection be confined to one side," the converse is true *only* when the obstruction has been confined to one side for a time and afterwards affects the other. I think facts show that it is not true when both kidneys have been acted upon similarly and for the same length of time.

Symptoms.—The previous history and the condition of this patient for some time after admission into hospital had reference to the disease of the bladder ; it was only during the last month of life that symptoms were produced by the secondary disease of the kidneys.

The blood and pus found in the urine from time to time, the former very frequently, and often in large quantity, must have come from the bladder. It is true pus was found after death in the substance of the right kidney, but the suppuration was too slight and too recent to account for pus in the urine several weeks before death. Hæmaturia has been noticed in many cases of hydronephosis, but is far from being an invariable symptom. It has been due generally either to injury inflicted by an impacted calculus ; or to rupture of blood-vessels in the congested cyst-wall, for both Hunter and Howship have demonstrated

the existence of numerous vessels and good-sized capillary arteries on the inner surface of the distended sac.[1] In the presence of a vascular growth in the bladder there is no need to look further to account for the hæmaturia in the above case.

The atrophy of the left kidney did not cause any serious inconvenience so long as the obstruction was confined to its own side, and the hypertrophied right kidney was allowed to perform duty for both; but as soon as the lower extremity of the right ureter was encroached on by the vesical growth the right kidney became involved. It was after this that pain in the right hypochondriac and lumbar regions, peristent vomiting, and constipation of the bowels, occurred. These symptoms have been noticed in most cases of hydronephrotic tumours. The only one requiring further comment is the constipation; this probably was caused by the pressure and dragging of the tumour upon the adherent bowel. No doubt too, in its turn, the loaded bowel gave rise to increased obstruction in the ureter and thus augmented the size of the tumour, and by pressure upon it provoked pain and sickness. It was noted on one occasion that the lumbar swelling disappeared after an enema and the discharge of a quantity of solid fæces. This feature, which by itself might be sufficient to lead one to a wrong diagnosis as to the nature of the tumour, has also been observed in other cases of hydronephrosis.

Treatment.—There is but little to say about the treatment in such a case. With one kidney in greater part destroyed, and the other partially obstructed and in a state of suppurative inflammation, life could not have been prolonged under any treatment; all the relief it was possible to afford was given by the removal of those portions of the vesical growths which had been the cause of so much pain in voiding urine.

The amount of pressure by the tumour of the kidney on the surrounding parts was never sufficient to suggest the

* *Vide* specimens in College of Surgeons' museum.

idea of tapping. As, however, this treatment had to be resorted to in many of the cases which have been published, I shall briefly consider the question as to the best way of emptying such tumours when assistance is necessary. An attempt might be made to overcome the obstacle to the passage of urine by friction. Two cases at least are on record in which the tumour subsided after manipulation. One was under Dr. Roberts in the Manchester Infirmary, the other was reported by Dr. Broadbent. In Dr. Roberts' case the tumour was rubbed in every direction with a lubricating ointment every other morning and after the third application the patient, a girl of eight years, suddenly passed a large quantity of urine, and the swelling forthwith subsided and did not return. In Dr. Broadbent's case a child three months old, whose abdomen had been large from birth, though urine had been passed in usual quantity, had an enormous tumour which disappeared after friction with iodine ointment. The child, however, subsequently died. Probably this treatment which answered so well in these two cases was suggested by a third case reported by Mr. Thurnam in 1837. A little boy four months old had a lobulated tumour from alternate dilatations and contractions of the whole length of the ureter; the mother of the patient was in the habit of rubbing it with her hand to relieve pain, and when she did so she noticed the child's bladder swelled up as large as an egg.

In many cases, however, manipulation could not be tried; either because the tumour is far too painful to allow of it, or because the degree of distension would make the risk of rupturing the cyst-wall too great. In these cases, as well as after the failure of rubbing, paracentesis is the only means of relief and then the place for puncturing must be considered. To rely on adhesions being formed between the tumour and abdominal wall in all cases would be unwise; and to puncture through non-adherent peritoneum would be likely to lead to extravasation of urine (or pus in cases of pyonephrosis) into the peritoneal cavity, an accident which has proved fatal in more than one case.

For this reason it would often be unsafe to operate on the front of the abdomen; moreover there would be the danger of puncturing intestine, which, as a rule, is in front of, and adherent to the sac of the tumour. On the other hand, care should be taken not to injure the substance of the kidney.

Under these considerations Mr. Joseph Thomson, of Nottingham, selected the interval between the last two ribs, near their anterior extremities (vide 'Path. Soc. Trans.,' vol. xiii), as the point at which to introduce the trocar, having previously made an incision through the integument and muscles. In this spot he punctured with success on three different occasions, in the same case, a tumour of the *left* kidney.

Mr. Spencer Wells says (' Dub. Quart. Journal,' vol. xliii, p. 128, 1867), " I have twice opened peri-renal abscesses in the loin, and in one case removed a small renal calculus through the opening; but the case just related " (No. 40 in my table) " is the only one in which I have punctured the kidney through the abdominal wall. It was clearly a hazardous proceeding, but the danger of rupture of the rapidly increasing sac into the peritoneal cavity appeared to be so great, and the suffering was so excessive, that tapping appeared to be less dangerous than expectation." The tumour was on the right side, it contained between two and three pints of pus, and fluctuated distinctly at a prominent spot near its middle where an aspiratory trocar was inserted. This spot was midway between the umbilicus and the anterior superior spine of ilium.

In a case reported by Dr. Cooper Rose the tumour is described as having presented a prominent point, " equidistant from the crest of the ilium and the umbilicus on the left side," where the skin was livid and the sac thin. The cyst was tapped, it is to be inferred, at this spot.

Again, in Dr. Hillier's patient the puncture was made " half an inch below and an inch and a half to the right of the navel." These cases might be thought sufficient to prove that there is no danger in puncturing far forward,

but it is clear in those of Mr. Wells and Dr. Rose either
that adhesions must have formed, or what is more pro-
bable that the peritoneum had been carried forwards so
as to leave the side of the tumour uncovered by it.

In the absence of any very distinctly fluctuating spot
where the skin is discoloured or the sac thin it would be
best to insert the trocar near the place proposed by Mr.
Thompson. If the tumour be of the left kidney, as in
his case, no better spot can be selected than one just
anterior to the last intercostal space; but if of the right
kidney this is too high, as the liver in all probability would
be traversed. I have on several occasions tested the truth
of this statement in the post-mortem room, and in only
one experiment did the liver escape; in this single in-
stance it was very small and markedly distorted from
cirrhosis. In one case, that of a young person with
healthy viscera, whose death had been caused by a fall, the
liver was considerably displaced by effused blood; but in
spite of its upward displacement it was transfixed by the
needle three quarters of an inch above its right free edge.

In no instance was the spleen punctured by inserting
the needle in front of the eleventh intercostal space on the
left side.

If there be no indication for operating elsewhere I
think the best spot to select when the tumour is of the
right kidney is one half way between the last rib and the
crest of the ilium, between two inches and two inches and
a half behind the anterior superior spine of the ilium.
This spot is on a level with the front of the bodies of the
lumbar vertebræ, and a needle here passed horizontally
inwards will be altogether in front of the kidney, and will

dilated pelvis of the kidney will be tapped at its anterior and lower part.[1]

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[1] Mr. T. F. Pearce, Junior Demonstrator of Anatomy at the Middlesex Hospital, made for me the following notes of some of the cases experimented upon. The results in every instance were much the same.

A probe being passed through the abdominal wall from within outwards, on a level with the anterior surface of the lumbar vertebræ, and crossing horizontally the lower part of the pelvis of the kidney, pierced the following structures:

EXPERIMENT I.—Male, 65. On the right side liver traversed and peritoneum punctured. On the left side intestines and peritoneum traversed. The external aperture is just above last rib, in an antero-posterior level, 2 inches behind the anterior superior spinous process of ilium.

EXPERIMENT II.—Male, 44. On the right side liver traversed. On the left side colon and intestines (small) traversed. The external aperture above twelfth rib, in antero-posterior level, 2½ inches behind anterior superior spine on both sides.

EXPERIMENT III.—Male, 44. On the right side liver traversed. On the left side intestines and peritoneum traversed. The external aperture opposite apex of eleventh rib, in antero-posterior level, 2 inches behind anterior superior spine on right side; but opposite apex of tenth rib, though on same antero-posterior level, on left side.

EXPERIMENT IV.—Female, 44. On the right side liver and peritoneum punctured. On the left side through intestines and peritoneum. External aperture between eleventh and twelfth ribs, in antero-posterior level, 2 inches behind anterior superior spine on both sides.

EXPERIMENT V.—Female, 56. On the right side liver with gall-bladder punctured. On the left side intestines and peritoneum traversed. External aperture between eleventh and twelfth ribs on right side, over eleventh rib on left; antero-posterior level similar to preceding case.

In both these cases, though the ureters were much distended, the needle passed over them. In the latter the right ureter was double at its upper portion for about 8 inches; it was much distended.

EXPERIMENT VI.—Male, 14. On the right side liver, &c., traversed. On the left side intestines and peritoneum traversed. External aperture just
. . . . nterior level, 2½ inches behind

rough the lateral wall of the
nth and twelfth ribs, and in an
superior spine of ilium, pierced

de liver (contracted, hobnailed)
ctured on both sides.
right side liver, with intestine,
tured on left side.
n, were traversed. In all, except
led, was punctured in one. The
e inch.

It is unnecessary to dwell upon the importance in all cases of hydronephrosis of relieving pressure from retained fæces by the use of enemata and purgatives.

Rarity of Intermitting Hydronephrosis.—The frequency of hydronephrosis as a pathological condition is well known and has been already referred to; its clinical existence as an abdominal tumour is undoubtedly rare. Still rarer is it for such a tumour to disappear spontaneously or to intermit.

I have put together in an appendix all the cases I have met with in which an abdominal tumour was observed at all (except those of congenital hydronephrosis where death occurred in utero or very shortly after birth); and it will be seen from this list that in only a few instances has there been any subsidence or intermission.

In six cases out of a total of forty-seven the tumour subsided *altogether* or nearly so, and did not reappear. In two cases already referred to it entirely subsided after friction. Of the rest (*a*) one was reported by Dr. Gintrac in which the tumour sensibly diminished after an abundant liquid motion, and then gradually disappeared altogether. The patient, a woman forty-eight years of age, died, and it was then found that a communication had been formed between the sac and the colon. It is not a very unusual thing in pyonephrosis for the sac partially to empty itself through some part of the bowel, most frequently through the colon. Such a case is mentioned by Howship, in which the tumour in this manner several times diminished though it never actually disappeared.

(*b*) Lamotte has described the case of an infant three days old, who had a large tumour in the abdomen and had passed no urine since birth, but the swelling entirely vanished after a membranous obstruction at the vesical orifice of the urethra had been broken down with a sound.

(*c*) In Dr. Hare's case, an abstract of which is given further on, there were two tumours, one of which intermitted and the other entirely subsided.

(*d*) Mr. James Johnson in 1816 published a remarkable

case in which the swelling first detected during pregnancy subsided some days after delivery and fifteen days before the patient's death. This case has been considered one of pyonephrosis rather than hydronephrosis, and such it most likely was, but Johnson, though he described the fluid as white like milk, seems not to have thought it was pus.

In only six cases is it stated that well-marked intermissions occurred. The first of these is referred to by Dr. Bright in his 'Clinical Memoirs on Abdominal Tumours,' in the following paragraph : "The third form of tumour arising in connection with the kidney is the simple distension of the pelvis and ureter with the natural secretion, owing to obstruction in the ureter or bladder. But one of the most striking cases I can at present recall has been published by my friend Mr. Estlin, of Bristol. In this instance the distended ureter produced a large tumour, which for some time excited much anxiety and great doubt as to its nature, till it was found that by a little pressure it was made to discharge its contents into the bladder, under which treatment it gradually contracted; but the ureter was found greatly enlarged and thickened after death, though it had not then been distended for a considerable time." Dr. Bright unfortunately did not give the reference to Mr. Estlin's case, but it is to be found reported, in part, in the 'London Medical Gazette,' vol. ii, 1828; and completed in vol. xx, 1837, of the same journal. The tumour was an enormous dilatation of the right ureter from obstruction caused by three semicartilaginous tumours of the prostate which formed a complete valvular impediment. No mention is made of the state of the right kidney, nor of the left kidney, nor of its ureter, and the inference therefore is that only the lower end of the right ureter was dilated. The tumour was pointed out by the patient himself many days after he first put himself under treatment. It was "of an oblong form, situated in the right hypochondrium, about the edge of the rectus muscle, extending nearly from the eleventh

rib to the right side of the symphysis pubis, and being particularly prominent about the situation of the internal abdominal ring. It somewhat distended the integuments so as to be perceptible to the eye and might be considered to be about three inches in width." Four days after it was first noticed a catheter was passed, three pints of urine were drawn off and the tumour disappeared. It formed and disappeared again and again, but always subsided after pressure and the use of the catheter; in fact, as there was no control over the bladder, the catheter was used every five or six hours, and thus the urine was not allowed to accumulate to any extent. In his first publication Mr. Estlin expressed the opinion that the tumour was a bulging of the inner coat of the bladder through the muscular layer, and thought it necessary to defend himself for not having arrived at this (as it turned out erroneous) conclusion earlier. Though the tumour was intermitting it cannot be called hydronephrosis, since it is not stated that there was any distension of the pelvis of the kidney. This, however, is only a matter of degree, for had life been prolonged, or the obstruction been at the orifice of the ureter instead of in the prostate, the pelvis of the kidney would have been dilated and the secreting structure absorbed, no doubt. This case, like Mr. Thurnam's, shows that a distended *ureter* may cause an abdominal tumour.

The second case, taken in chronological order, which I have been able to find is quoted by Dr. W. C. Roberts, of New York, in a paper " On Diseases of the Fœtus," in the ' American Journal of Medical Sciences ' (1841, ii, 391). " A lady of Philadelphia gave birth to a healthy child; two weeks afterwards, the child having made no water for two days, a large fluctuating irregular tumour was found, occupying the lumbar and iliac regions, which disappeared next day after a copious flow of urine. This occasionally recurred, but when eighteen months of age the child died of long-continued irritation. The ureters were found enlarged to nearly double the size of

the large intestine, and completely distended with urine. At their pelves they partially covered the kidneys, at their entrance into the bladder they were small, and their valvular structure was entirely preserved. The bladder was larger than natural, but bore no appearance of inflammation." Nothing is said as to the state of the kidneys.

Sir Henry Thompson exhibited, for Mr. Joseph Thompson, at the Pathological Society, in November, 1861, an enormous tumour connected with the kidney which had been removed from the body of a patient of the latter gentleman. The man first sought advice in May, 1851, having suffered with symptoms of stone in the kidney for some considerable time previously. There was enlargement of and tenderness on pressure over the left hypochondriac, lumbar, and iliac regions; after passing, all at one time, more than a " pot-de-chambre " full of water of the colour of port wine he was suddenly relieved. From the end of November in the same year till the end of January, 1852, the same symptoms recurred and went on increasing in severity. The side was greatly enlarged, very tender and obscurely fluctuating; and the sufferings of the man were very considerable. The tumour was therefore tapped and eight quarts of dark coloured urine were drawn off and soon afterwards, it is stated, " the further contents of the sac flowed in the natural direction along the ureter. This could be ascertained from the quality and quantity of the urine." In December, 1852, it was necessary to tap the cyst again, which from the first had been supposed to be the dilated pelvis of the kidney. The patient got well and did not require the operation again till March, 1860, when seven quarts of fluid were taken away, not long after which the fluid again flowed *per viam naturalem*, and the patient remained well until September, 1861. The disease then progressed, as on former occasions, for twelve days, when the contents were extravasated into the peritoneal cavity through an ulcerated opening on the fore surface and towards the left side of the sac. Through

this opening the finger detected a loose, hard, calcareous body. The ureter entered the sac very obliquely at its lower part and anteriorly, and it was thought by Mr. Thompson that this position and obliquity of the ureter accounted for the closure of that tube, by pressure, when the sac was full, and the open state of it when the sac was empty.

The most extraordinary case of intermitting hydronephrosis on record is, I think, that reported by Dr. Hare in the 'Medical Times and Gazette' (pp. 29, 233, and 258) of 1857. A married woman, aged 38 years, first began to have pain in the left loin when twelve years old, and suffered from it for some years. At the age of twenty-eight pain attacked the right side of the abdomen and the loin of the same side; and with the exception of one year she was seldom afterwards free from pain. At times the pain was very severe, and then a large tumour was felt on the right side of the abdomen, which subsequently disappeared; the tumour again appeared and disappeared on several occasions. In 1855 a large tumour was also discovered in the left side of the abdomen; this disappeared some time before death and did not again become evident. Death occurred with symptoms of uræmic poisoning. On post-mortem examination very considerable double hydronephrosis was found to have existed; the right kidney and its pelvis were still distended, the left was almost empty. Dr. Hare was inclined to the belief that the cause of the obstruction was a congenital one; the only detectible obstacle to the flow of the urine was a twist in each ureter upon its own axis. As soon as the ureters were dissected up a little from the tissues surrounding, and the coil untwisted, the urine which had accumulated in the right side gushed out freely. The coincidence of the two kidneys being so similarly yet so peculiarly affected is, indeed, as Dr. Hare says, truly remarkable. The patient first came under Dr. Hare's notice in 1854.

Dr. Hillier, in the 48th volume of the 'Transactions of the Royal Medical and Chirurgical Society,' refers to a

case under his observation in 1855. It was that of a married woman who for three or four years had been subjected to attacks of pain in the right side of the abdomen attended with an oval swelling in the right lumbar region which disappeared as the pain subsided. During the height of the attacks the urine was very scanty, but afterwards was very abundant and turbid for some days. The patient was alive ten years afterwards and had not had entire suppression since 1855.

The most recently reported case I have met with formed the subject of a paper by Dr. Thomas Cole, of Bath, which was read before the Bath and Bristol Branch of the Medical Association, and is reported in the 'British Medical Journal' of September, 1874. A young man, æt. 23, who was first seen by Dr. Cole in 1873, had enjoyed good health till three years previously, when he was seized with pain in the left lumbar region, accompanied by sickness. It lasted a few hours. The attacks recurred at intervals of one or two months. Latterly the intervals had been shorter, not more than a fortnight; and the attacks lasted from a few hours to several days. The bowels were generally confined. Never noticed anything wrong with his urine, which on examination contained nothing abnormal except a superabundance of octahedral crystals of oxalate of lime. During 1873 the intervals varied from one to seven weeks, showing a general tendency to become less and less frequent. A tumour was felt in the left side of the abdomen very shortly after the commencement of each attack, which distinctly pushed the belly of the left rectus muscle forward, and altered the contour of the front surface of the abdomen. There was obscure resonance in front of the tumour. Fluctuation was not satisfactorily made out. The tumour was easily moved about, and could be made to descend one or two inches by a deep inspiration. During the attack the urine decreased to $\frac{3}{4}$ pint, in the intervals it varied from $1\frac{1}{2}$ pints to 3 pints; when the swelling passed away the quantity was increased to $1\frac{1}{2}$ pints in the day, and was voided very frequently—

three times in an hour or so. As a rule the tumour went away very rapidly, for instance, on one occasion the swelling was as large as ever at 11 p.m.; at 4 a.m. the patient awoke and found it gone, and at 10 a.m. Dr. Cole could find no trace of it. The patient is still under treatment, but the diagnosis is that a calculus of oxalate of lime is lodged in the pelvis of the kidney, and occasionally causes obstruction by falling over the entrance to the ureter.

A notable symptom in this case, and one I do not remember reading of in any other, was the large quantity of liquid *expectorated* during the attack, which had the characters of a mixture of saliva and gastric juice. More than a pint and a half of this was expectorated during one attack.

Dr. Cole has been good enough to give me information about his patient up to the present time, and on February 15th, 1876, he wrote: "Some months ago my patient while on a visit to London was seized with one of his attacks after an interval of nearly a year. It was severe, and up to this time the swelling, which always subsided before, had not disappeared. He suffers no particular pain and seems fairly well in health, but there is the swelling and I fear it has become permanent. Sir W. Jenner saw him during the illness, and came, I believe, to a similar conclusion to mine; at all events diagnosed a kidney tumour."

I am indebted to Mr. Bryant, of Guy's Hospital, for the following notes of an unpublished case which he saw in consultation with Dr. Emanuel May.

Mrs. S—, æt. 47, of 25, —— Street, Regent's Park, when twenty years of age had a swelling in the abdomen which subsided spontaneously after she had passed urine of a coffee-ground character.

Soon after the disappearance of the tumour, which had been considered to be ovarian, the lady was married. She remained well, and had two children between her marriage and eight years ago, when she was attacked with typhoid

fever. Then the abdominal swelling recurred and a tumour, which steadily increased in size, formed in the left loin. Her general condition associated with these symptoms caused alarm, and, in fact, Mrs. S— became very ill indeed, and was not expected to live; but at the expiration of fourteen weeks a copious discharge of blood and pus took place from her bowels and continued for several weeks. Ultimately she quite recovered from this illness, the tumour disappeared, and the abdomen returned to its normal dimensions. Again she remained well for a long time, but on the 18th of May, 1874, pain and swelling occurred once more in the left loin. Six days later there was a soft tumour above the left iliac fossa which was thought to be faecal.

On June 2nd the swelling extended from the ribs to the crest of the ilium and was tense though elastic; the urine was of a very high colour and looked as if it had been mixed with bile. A few days later the tumour, which was then supposed to be " suppurating hydronephrosis " of the left kidney, reached as far as the umbilicus.

On June 6th Sir W. Jenner saw the patient and suggested that the tumour should be tapped; this was accordingly done and ninety ounces of offensive pus were drawn off by Mr. W. Adams, of Harrington Square, in whose practice the case occurred. The operation was followed by some improvement, but on the 16th of June the sac refilled and three pints of foetid greenish fluid were removed with the aspirator.

On June 28th the urine for the first time contained pus as well as blood.

On July 3rd the tumour had again refilled; Mr. Bryant was then called in to see the patient and laid the sac freely open by an incision such as is made for the operation of colotomy. The kidney was felt by the fingers introduced through the wound, and a quantity of offensive purulent fluid mixed with lymph and broken-up tissue was evacuated. Considerable relief was given by the operation, but death from peritonitis took place ten days later. A post-mortem

examination was made, but I have not been able to obtain the details of it.

It would be found upon inquiry, I think, that pyonephrosis has given rise to an abdominal tumour much more frequently than hydronephrosis. In searching for cases of the latter kind only, to tabulate them for an appendix to this paper, I have passed over all which were throughout unquestionably cases of pyonephrosis.

In some instances, however, the abdominal tumour, which at a later period was pyonephrotic, seems not to have been so at an earlier date. These I have included in the subjoined list. The case last quoted was no doubt one of this kind, as the swelling subsided entirely on one occasion without the escape of any fluid other than blood and urine.

In conclusion, I would suggest that in all probability a great number of the tumours which, having been called *ovarian* upon good authority, have, nevertheless, vanished spontaneously, were hydronephrotic in nature and disappeared by a normal process through a normal outlet of the body. It is a noteworthy fact, and one showing the liability to error in diagnosis in such tumours, that of the twenty-three cases included in the first part of the subjoined table twelve occurred in females, and of the twelve women no less than seven were regarded as suffering from ovarian disease; while of these seven five were submitted to the operation of abdominal section on the strength of this wrong opinion.

Mr. Wells also, in a table of cases supposed to have been ovarian " in which an exploratory incision was made," includes two of renal cyst. In each the cyst was " exposed, tapped, and drained." Both patients recovered and remained well for some time afterwards. These two cases are not included in the following appendix because the nature of the cyst, whether hydronephrotic or otherwise, is not mentioned.

Note to Appendix.—In the course of the discussion on this communication Dr. Hare referred to two other cases which had been under his care, in each of which hydronephrosis had given rise to an abdominal tumour. In each case the patient was a young woman ; in each the tumour intermitted ; in one it was on the right side, in the other on the left.

In April of this year my attention was drawn by Dr. Fagge to a case in the clinical ward at Guy's Hospital, under his care, and to his kindness I am indebted for the following note of it.

A married woman, æt. 37, the mother of five children, had a globular tumour six inches in diameter situated in the left lumbar and iliac regions. Just previously to her last confinement, about two years before admission, she had felt aching pains in the left side of her abdomen which, however, were not continuous. The tumour had only been detected six weeks; it was moveable, not very tense, and there was resonance in front of it. It was neither painful nor tender. It fluctuated. The urine, dark at times, was generally clear and without any deposit; sp. gr. 1022, acid ; it contained neither albumen nor sugar. The tumour was tapped on May 1st, and forty-five ounces of a limpid opalescent fluid were withdrawn. This fluid was odourless, neutral, contained no hooklets ; a few epithelial cells were seen under the microscope. It gave no reaction with heat and nitric acid, nor with Pavy's solution. Its specific gravity was 1004. Some of it was evaporated to dryness ; scarcely any residue was left, and what there was consisted of sodium salts. Tests for urea and uric acid gave no reaction whatever. The patient left the hospital on May 20th, 1876. Subsequently to the tapping a round hard substance could be felt in the left loin, in the situation of the kidneys.

This tumour was considered to be, and no doubt was, of hydronephrotic nature. The cause of it was not ascertained.

It is very noteworthy that the fluid did not present the

normal characters of urine, but in fact consisted of water with a large quantity of chloride of sodium and a few cells.

Such clear limpid fluid-contents have been found in other instances. Sir James Paget has mentioned to me a case he had seen in which the fluid contained in the cyst was very pale and very unlike ordinary urine in odour, colour, and other characters.

I have referred to this point and have suggested an explanation of it in a communication on 'Congenital Hydronephrosis.'

Cases of hydronephrosis in which an abdominal tumour was formed.

A.—Those in which the tumour never spontaneously subsided or intermitted.

No.					Remarks and post-mortem examination.
1	Lancet, 1861, vol. ii, p. 472	Dr. Farre	A man, æt. 46, who for years had been subject to pains in the abdomen, had been ill for five months, when a tumour was discovered in the right side. The tumour was several times tapped, and iodine was injected. Death occurred in eighteen months	The tumour filled up after each tapping. It never subsided except as the result of tapping	Supposed to be a hydatid tumour of the liver. A calculus impacted in the ureter had led to sacculation of the kidney and distension of its pelvis.
2	Philosophical Transactions, 1747	Mr. Samuel Glass	A woman who was born dropsical lived to be 23 years of age. She was well proportioned, except for the enormous size of the abdomen, which had been disproportionate all her life. The only symptom was pain on passing urine. The tumour was on the right side. The abdomen at the time of death was 6 feet 1 inch in circumference, and from xiphoid cartilage to os pubis it measured 4 feet ⅜ inch	No subsidence or diminution of tumour	The tumour was due to distension of the right kidney; it contained 30 gallons of light-brown limpid fluid. The ureter of this kidney was abnormal in course, and opened very obliquely into the cavity of the sac. It then passed upwards and to the right for twelve inches, whence it was deflected downwards.
3	Gœttingen gel. Anzeigen, 1777, s. 1196	Haller, quoted by Bayer, vol. iii, p. 496	A woman who for years had suffered from dropsy had had also for several years a tumour below the umbilicus	No variation in size of tumour mentioned	The tumour was formed by one of the kidneys, which had become degenerated into a membranous sac, white, and filled with water. The other kidney was quite healthy and in its proper position.
4	Medical Commentaries, vol. ix, p. 282, 1785	Mr. Phillip Martineau, of Norwich	A man was admitted into Norwich Hospital with a large tumour on the left side of the abdomen, which fluctuated. Two years previously he had been in hospital for the same complaint, and had been tapped. Till within two or three months of readmission he had remained well. The encysted tumour was now punctured with a lancet, the fluid escaped into the peritoneal cavity, and death resulted	No subsidence of tumour except after tapping	The left kidney was dilated into a cyst large enough to contain eight pints. The cause of obstruction was not discovered. Mr. Martineau "knew of no other case except by Lieutaud."

Remarks and post-mortem examination.
The right kidney had no trace of its natural structure, and its pelvis terminated in a *cul de sac*. The ureter was natural towards the bladder, but near the kidney it had the form of two impervious cords, which became more sub-divided, and were applied to the kidney in the form of a goose's foot.
The cyst contained more than 60 lbs. weight of a fluid, rather aqueous than urinous. There remained nothing of the kidney but its capsule.
The left kidney was transformed into a pouch containing a great quantity of fluid paler than normal urine. The ureter was so narrowed that a pin was introduced with difficulty. The right kidney was larger than usual.
The right ureter was plugged by a calculus.
The right ureter was compressed by indurated and enlarged lympha-tic glands situated between the uterus and the iliac muscle. The left kidney and ureter were un-affected. The fluid in the cyst was yellow and without odour.

No.	Reference	Author	History	Course	Remarks	Post-mortem appearances
10	Traité des Maladies des Reins, tom. iii, p. 490	Bayer	A man, æt. 64, having been seized with pain all over the abdomen, but especially severe about the left kidney, was compelled to keep his bed. No urine was voided, nor was there any desire to micturate for ten days. The bladder was not distended. There was a large tumour, which stretched obliquely from the right hypochondriac towards the left iliac ... to be distended right kidney. The patient died in less than a month from the commencement of this attack. At the age of 22 pain had been felt in the region of the right kidney, which extended along in the direction of the right ureter, and blood was passed by the urine. Little by little he improved, and remained well for several years. For fourteen years before the final illness the abdomen had been increasing	Continual increase in size of tumour	Upon this case Bayer remarks that the alteration of the right kidney accounts perfectly for the old illness b during life for the cause of death, which was inevitable when the left kidney (the only one excreting urine) became involved	The right ureter was completely plugged at one spot by an impacted calculus. Above the obstruction the ureter was dilated, below it was of the ordinary measurement. The left kidney was swollen and red, and its pelvis was notably dilated; the left ureter contained a small calculus about five inches from its upper end.
11	Arch. f. d. Hollandische Beitr. z. Natur und Heilk., Bd. i, p. 196	Boogard	A young man, æt. 20, had suffered from time to time from vomiting; a doubtfully fluctuating swelling was detected in the right flank; death occurred five days afterwards	No subsidence		The right kidney was affected, the left was natural. The right renal artery divided into two branches, the lower of which compressed the ... pper ... ent.
12	Wiener Med. Halle, 1864, p. 139	Prof. Dumreicher	A girl, æt. abdomen? ... ling in the grew enormously and uniformly distended the belly. It was frequently tapped and injected, but always refilled.	No subsidence except as the result of tapping		The right kidney was the one which formed the tumour. The left was hypertrophied. The cyst was ... and ... these into loculi. The cause of the obstruction was not clearly made out. It was considered to be congenital, and due to obliquity of the ureter.

No.	Reference.	Authority.	Brief abstract of case.	Variations in tumour.	Remarks and post-mortem examination.
13	Langenbeck Archiv f. Klin.-Chir., 1865, Band vii, p. 219	Krause	Hydronephrotic cyst mistaken for ovarian. Extirpation was attempted. Death next day. The tumour had been tapped one year before under the belief it was ovarian	No subsidence	The kidney was entirely atrophied, and its pelvis enormously distended. The cause of obstruction was a valvular flap of membrane at the renal orifice of the ureter.
14	Berlin Klin. Wochenschr., vi, p. 23, 1869	Prof. Simon's Clinic, Heidelberg	A hydronephrotic tumour was punctured with trocar and cannula, and the cannula was left in situ. Death from peritonitis followed twenty-two days after the operation upon what was considered to be an ovarian cyst	No subsidence before tapping	The ureter was not permeable, but the cause of its closure was not explained by post-mortem examination.
15	Path. Soc. Trans., vol. xiii, p. 151	Dr. W. J. Little	A male, of 6½ years, had at 2 years been treated for ascites, and was relatively as large in the abdomen as a woman at the full period of gestation. He was born with a large belly, which had gradually increased to this condition. Paracentesis had been performed at 8½ years of age. Limpid fluid was evacuated. Seve rately a fistulous opening was established. The nature of the case was diagnosed before death	No subsidence except as a result of tapping	The right kidney formed a large cyst. The left was also mooniated. No explanation of the origin of the disease.
16	On Urinary and Renal Diseases	Dr. Roberts, of Manchester	A woman who was supposed to be suffering from ovarian disease, and who had been twice tapped under this idea, died of peritonitis after the second tapping	No subsidence except as the result of tapping	The right kidney and its pelvis were monstrously dilated. The ureter opened so obliquely into the pelvis of the kidney as to be quite valve-like.
17	Wurt. Correspondenz Blatt, Bd. xii, p. 266	Faber	A little boy, 5½ years, who had been ventricose from birth, fell from his chair and died suddenly from the injury received thereby	No subsidence	Both kidneys and their ureters were distended. No cause mentioned.

18	Glasgow Med. Journal, vol. ii, 1867	Dr. A. R. Simpson	While making experiments in acupressure, the body of a woman, æt. 50, was supplied, who had complained a few days before her death of an abdominal tumour. It was regarded as an ovarian tumour, but as it caused no uneasiness no special examination was made of it during life	No subsidence	The right kidney formed a cystic tumour as large as a fœtal head. The cause was a thickened band of fibrous tissue which passed along the brim of the pelvis from the promontory of the sacrum and compressed the right ureter. The left kidney was hypertrophied.
19	Edin. Med. Journal, vol. xiii, p. 468, 1867	Dr. Bryce, of Dalkeith	A large tumour occupied the right lumbar and iliac regions of a unmarried woman. Towards and ... was confined to within ... the le ... in two inches of the umbilicus. The tumour in the right side was always spoken of by the patient as being like "a river in her side." Death occurred within a short time after delivery, and the tumour of the right side did not subside before that event. The patient was seen during life by Sir A. Simpson. The p.-m. examination of the body was made and reported on by Dr. A. R. Simpson	No subsidence	Left kidney pale and flabby, but increased in size. Right kidney immensely distended into tumour, which extended from the lower surface of liver to pelvic brim. The colon was adherent to tumour, so was the right ureter. The latter was pervious and slightly dilated, and bent upwards on the cyst very suddenly. After dissecting off the ureter from the cyst 96 ozs. of healthy, highcoloured urine of sp. gr. 1011 flowed away through it.
20	Path. Soc. Trans., vol. xxv, p. 174, 1874	Drs. Woodman and Cayley	A male child, æt. 5 months, had from birth a larger abdomen than normal. After three months it increased, and the child passed but little water ... occupied th ... to the liver ... were said to be contained in the 4 oz. of blood-stained fluid (sp. gr. 1010) which were withdrawn. No urea in fluid. Three other punctures at intervals of 6—12 days allowed of more blood-stained fluid being removed. This fluid contained urea, but no traces of hydatid. Death from broncho-pneumonia occurred a few days after the last tapping	No subsidence except as the result of tapping	l into ... ermed ... elvis, ... ed by ... reter ... had been cut off and was not examined. The hydronephrosis was thought to have been associated with echinococcus cyst, but no proof of this was found on postmortem examination.

No.	Reference.	Authority.	Brief abstract of case.	Variations in tumour.	Remarks and post-mortem examination.
21	Archiv f. Gynäkol., 1870	Dr. Schetelig	An enormous hydronephrotic tumour was removed under the idea that it was ovarian. Death. The left kidney was the one affected	No subsidence	—
22	Dublin Quart. Journal of Med. Science, Feb., 1867; also Diseases of the Ovaries, pp.208 et seq.; also Path. Soc. Trans., vol. xviii, p. 167	Spencer Wells	A married woman, æt. 43, "suffered much from the urine" from her fourteenth year. At this time she was felled to the ground by a blow on the abdomen with an iron shovel, and for eighteen months subsequently she was ill. For years afterwards she suffered pain and distress, which she referred to this injury. All her married life (28 years) she repeatedly suffered (, the ber) abdomen, and her pr were always attended with distress. When 39 years of age she had had a swelling in the ... to be ... in the ... fines albæ, when two gallons of dark fluid were withdrawn. Five months later she came under Mr. Wells, and soon afterwards was operated upon for ovarian tumour	No subsidence except as the result of tapping	The right kidney was enlarged and were ... sac had cal- f one calyx. The bladder was contracted, the uterus and ovaries were healthy. The left kidney formed a cyst larger than an adult head. The stroma was in fact atrophied, in part hypertro- spleen, pancreas, tomach, duodenum, coils of small intestine, and other structures were very firmly adherent to the cyst wall. No cause mentioned of obstruction on this side.
23	Diseases of the Ovaries, pp. 216 et seq.	Spencer Wells	A girl, æt. 16, fat and of florid complexion, had found her abdomen increasing, since her ? to attract seized with pain on the right side. When she was admitted into the Samaritan Hospital fluctuation was distinct over the lower part of the belly, and the movements of a cyst were dis-	No subsidence before operation	Death on fourth day after operation from uræmia. The left kidney was so atrophied as to be almost useless. The right kidney was in great part converted into a cyst which held 12 pints. This case is very remarkable from the rapid and great elevation of

B.—*Cases quoted in the text in which the tumour intermitted.*

24	Medical Gazette	Mr. Estlin	*Vide* text.		
25	American Journal of Medical Sciences, "On Diseases of the Fœtus," 1841, ii, 591	Dr. Roberts, of New York	A healthy male child, æt. 2, seized with retention of urine. A large, fluctuating, irregular tumour was formed, occupying the lumbar and iliac regions, which disappeared next day after a copious flow of urine. This occasionally recurred. Death at eighteenth month	Intermitted on several occasions	The ureters were enlarged to nearly double the size of large intestine, and completely distended with urine. At their exit from the pelvis they partly covered the kidney. Cause of obstruction not mentioned.

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For several hours before death
the temperature exceeded 111°.

No.	Reference.	Authority.	Brief abstract of case.	Variations in tumour.	Remarks and post-mortem examination.
26	Path. Society Trans., vol. xiii, p. 128	Sir H. Thompson, for Mr. Joseph Thompson	Abstract given in text	The tumour subsided and remained well for nine months; it refilled, and did not again subside except after tapping.	Vide text. The peculiarity of this case is the long temporary relief given by the tappings.
27	Med. Times, and Gazette, 1857, vol. i, pp. 27, 233, and 258	Dr. Hare	Vide text.		
28	Med.-Chir. Trans., vol. xlviii, p. 86	Dr. Hillier	Vide text.		
29	British Med. Journal, 1874	Dr. Cole, of Bath	Vide text.		

c.—Cases quoted in the text in which the tumour subsided entirely.

No.	Reference.	Authority.	Brief abstract of case.	Variations in tumour.	Remarks and post-mortem examination.
30	'Urinary and Renal Diseases'	Dr. Roberts, of Manchester	A little girl, æt. 10. The tumour was shampooed three times; after the third manipulation she suddenly passed a large quantity of urine, and the tumour subsided	Subsidence after friction	
31	Path. Soc. Trans., vol. xvi, p. 164	Dr. W. H. Broadbent	Tumour in a female child, between 3 and 4 months old	Subsidence after friction	Both kidneys were affected. The obstruction was due to valves of mucous membrane near pelvis of each kidney. Urethra not examined.

32	Sydenham Soc., Bien. Retrosp., 1867 and 1868, p. 176	Dr. Gintrac	A woman, æt. 48, began (1865) to suffer from vomiting and alternate diarrhœa and constipation. In 1866 she was worse, and a tumour was observed in her left flank. The urine was clear and healthy on February 7th. On the 20th, after abundant liquid stools, the tumour sensibly diminished, and then gradually disappeared. The patient died	Subsidence of tumour by escape of urine per rectum	The secreting structure of the kidney was nearly destroyed. No calculus was found. The ureter was healthy.
33	Traité des Accouchements, liv. 1, ch. xxiv, 128	Lamotte	*Vide* text.		
27 e. s.	Medical Times and Gazette, 1857	Dr. Hare	This is the same case in which the tumour formed by the right kidney intermitted, and therefore it is referre... also. The tumour s... sided on one occasion and did not refill	*Vide* text.	
34	Medical Chirurg. Journal and Review, vol. ii, 1816	Mr. James Johnson	This case is included under those hydronephrotic cysts which subsequently suppurated. The tumour *almost* disappeared on one occasion, nine days after confinement and fifteen days before death of patient		

D.—*Cases in which the tumour diminished but never subsided.*

No.	Reference.	Authority.			
35	Path. Soc., vol. xiii, p. 187	Dr. Dickinson, for Dr. C. Hastings	A woman, æt. 70, had had for twelve years a tumour in the left hypochondrium, which at length filled the abdomen. She occasionally passed "nasty stuff" by the urethra, and the tumour then diminished somewhat in size. The tumour had been diagnosed ovarian.	Although the tumour is said to have diminished, it never subsided. In spite of occasional variations, there was, on the whole, such a progressive increase that the whole belly was at last filled by the tumour	The left kidney was converted into a sac full of a colloid or gelatinous substance. No obstruction found in ureter. Tumour probably caused by a calculus at some time impacted.
36	'Urinary and Renal Diseases,' ed. ii, p. 468	Dr. Roberts, of Manchester	A male, æt. 20, had from the age of 2 years been subjected to attacks of constipation, which became more frequent and severe. During the attacks the belly was swollen and tender. When under Dr. Roberts in 1867 an attack was closely observed. During it the urine was scanty, the abdomen was distended, and in each loin there was a bulging. No actual and defined tumour, but a general swelling of abdomen. Patient partially recovered from this attack, and died in another very shortly afterwards. Diagnosis made out accurately	It is stated that after copious discharges of urine and relief of intestinal obstruction, there was a sensible diminution in the elastic swelling on the left side	Both kidneys affected. The left was the larger cyst of the two. The upper end of left ureter was contracted, and opened so obliquely into the pelvis of kidney as to form a valve-like obstruction. Contraction congenital. On right side distension was due to an abnormal branch of the renal artery.

No.	Reference	Author			
87	Lancet, 1863, vol. i, p. 817. Med. Chir. Trans., vols. xlviii and lii	Dr. Hillier	Patient was born with a large abdomen, which simulated ascites till he was 4 years old. The swelling was then diagnosed as being a cyst of the right kidney. For a long time treatment for ascites was employed. After the tumour was rightly diagnosed it was frequently tapped	There was occasional reduction in the size of the tumour. The abdomen varied from twenty-four to twenty-nine inches before the first tapping	Death from acute tuberculosis. The cyst contained eighty-three ounces of pale lemon-coloured fluid. There was marked contraction of the lower end of the right ureter. The left ureter and pelvis were also slightly dilated, and contained some calculus and matter.
88	'Diseases of the Urine and Urinary Organs,' ed. 1823, p. xlviii	Howship	A young lady, æt. 24, first observed an enlargement of the abdomen in 1816, which slowly increased till it equalled an eighth month pregnancy. The tumour continued, and on November 5th, 1818, she first had retention, and the catheter was daily used till November 10th, when the urine declined from two quarts to a few ounces, and so continued till December 8th. Then influx took place and lasted twenty days, during which over seventeen gallons of urine were drawn off with the catheter. In May urine passed by the bowels, and then afterwards alternated, passing sometimes by the bowel and sometimes through the ...	There were occasional variations in the size of the tumour, but no complete subsidence	This case was not followed to the end. It was under Mr. Heaviside.
89	London Med. Gaz., vol. xx, 1887, p. 717	John Thurnam	A male child was born with tumefaction of the lower part of the abdomen, especially of the right side. It looked like two or three bladders. No urine passed till the third day after birth. The mother used to rub the abdomen to relieve pain, and in doing so she noticed that the bladder would swell and rise up under her hand to size of a large egg. The left ureter chiefly formed the tumour, but the left kidney was very large and its ...	Frequent alternations in size followed friction on the surface of the abdomen	The right kidney was completely atrophied, not being more than quarter of usual bulk, and showing no trace of division into cortical and medullary parts. There was ... right ... capsule. T ... way from ... sented by a thin impervious cord for about one inch, but the vesical ...

No.	References.
40	Traité des Maladies de Reins, tom. i p. 492
41	Ditto, p. 495

The tumour subsided to three quarters of its size

The right kidney formed a soft membranous mass eight inches long and five wide. It contained nodes, a quantity of fluid, four large calculi.

The remains of the left kidney could not even be found except by tracing carefully along the left ureter. It was only represented by a small membranous sac, which contained some fluid and a calculus.

Retention of urine occurred, and the catheter was required for five days on one occasion. After this attack the tumour diminished somewhat; death occurred two days later

Double hydronephrosis in a woman, æt. 41, was occasioned by renal calculi. Two years before the death of the patient there was found a tumour in the right flank. Patient died of what was called "malignant fever," produced by violent grief

contents of the tumour became purulent, i. e. the hydronephrotic cyst became pyonephrotic.

he obstruction was due to a rough calculus, which was lodged in the pelvis of the left kidney.

A married woman, æt. 27, died in the for Women after having been tap times. Swelling in left loin beg months after confinement. First bl thick yellow matter, and afterwe
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Variations in tumour.	Remarks and post-mortem examination.
No variations in size mentioned, except as the result of tapping	Tumour was formed by the dilated left kidney, which was firmly adherent to parts around. The ureter was dilated for an inch and a half, and then suddenly became contracted so as not to admit the smallest probe. No calculus. The cyst communicated with the adherent intestine.
The only change up to the time when the tumour was tapped was one of increase in size	The kidney, which was closely adherent to the supra-renal capsule, formed a large cyst containing thin ochrey matter and shreds of bloody pus; the tubular tissue had entirely disappeared, whilst the cortical part was reduced to a pale thin stratum. The ureter had degenerated into a hard thin cord and was entirely closed up.
The tumour increased up to the time of tapping, and subsequently refilled. Ultimately it completely disappeared, and the	This patient was well seven years after the first tapping. The case is related also in Mr. Wells' work on 'Diseases of the Ovaries,' p. 205, et seq.

No.	Reference	Author	Clinical history	Result	Post-mortem appearances
47	'Diseases of Urinary Organs,' p. 202, ed. 1833	Howship	Although the fluid which flowed through the sinuous opening was sometimes clear and urinous, it is not certain whether this was a case of hydronephrosic distension at first, or whether ... or motion. The urine was once or twice slightly bloody, but was never purulent	sinus quite closed up	Three pints of milky fluid were contained in a large tumour which occupied the left lumbar region. The kidney structure was entirely destroyed. A calculus blocked the upper orifice of the ureter.
34 e. s.	Medical Chirurgical Journal and Review, vol. ii, 1816	Mr. Johnson	A woman, æt. 40, in eighth month of pregnancy, presented two abdominal tumours ... h was the distended ... y and in... ovarian, ... nine days ... ntity of ... Death occurred fifteen days later. Symptoms had existed two years before the tumour was noticed	been No change in tumour is described. The pain ... sure ... One nearly complete subsidence, without subsequent reappearance	The right kidney formed a very large cyst adherent by very firm and old adhesion to the liver, small intestines, and psoas muscle. This cyst contained three pints of milky fluid. The ureter was dilated, but not obstructed. The cyst had probably held about five or six quarts of fluid. The bladder contained ... of similar fluid, ... chemical ex- ... made.

ON

"ATHETOSIS" AND POST-HEMIPLEGIC DISORDERS OF MOVEMENT.

BY

WILLIAM R. GOWERS, M.D.,

ASSISTANT PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND TO THE
NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC.

SINCE Dr. Hammond described in 1871 [1] certain forms of unilateral motor disturbance to which he gave the name of "athetosis," only two or three similar cases have been recorded, and in no instance has there been any improvement in the special symptoms. I have therefore ventured to bring before the Society two cases in which a closely analogous, if not identical, condition is or has been present, one of which is further interesting from the circumstance that the characteristic symptoms have, under treatment, almost entirely disappeared. I have added a brief comparative outline, illustrated by cases, of other more familiar forms of spasm to which athetosis is, I think, very closely allied.

The cases described by Hammond were characterised by an affection of hand and foot, by which the fingers and toes were in continuous *slow* movement, leading to curious distorted postures. The contraction was most marked in the hand, the fingers being variously extended and spread out, or partly flexed; the foot was inverted, the toes over-

[1] 'Diseases of the Nervous System,' p. 654.

extended. No motor weakness was recognised; the movements were to a slight extent only under the control of the will, and the disease was named from the inability to keep the fingers in a fixed position. Other symptoms associated in some cases were epileptiform attacks at the onset, tremor of tongue, and numbness of the affected side. In one case it is noted that the movements continued during sleep, and it is implied that this was the case in all. Other similar cases have been recorded by Clifford Allbutt, Currie, Ritchie, Fisher (Boston, U. S.), Gairdner; and Dr. Claye Shaw has called attention to the analogous condition which is sometimes seen in the limbs of imbecile children.[1]

The condition of athetosis which Dr. Hammond depicts possesses obvious points of resemblance to the condition of slowly-changing spasm occasionally seen after hemiplegia in early life, and more rarely after hemiplegia in adult life,[2] and the question occurs, are the two conditions essentially distinct? Dr. Hammond does not expressly distinguish the two, he merely alludes to the diagnosis of athetosis from "cerebral hæmorrhage,"[3] and gives as the differences that in the former (athetosis) the disordered movements continue during sleep, and in the latter (cerebral hæmorrhage) occur only on voluntary movements. But Dr. Hammond claims as instances of athetosis cases in which, as in that of Dr. Clifford Allbutt, the movements

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[1] Since this paper was read Dr. Martin Bernhardt has published ('Virchow's Archiv,' Bd. lxvii, Heft 1) a case which he regards as one of "athetosis." Its details differ much, however, from those which Hammond described, especially in the fact that the patient's hand was perfectly still when his attention was not directed to it, and that the movements were "tolerably quick." The case resembles much more those of quick, clonic (choreoid) spasm described further on, such as Case 13 appended. This may explain his conclusion that "athetosis" is only a unilateral chorea. The slowness and constancy of the movements in athetosis (as described by Hammond) certainly distinguish it from any movements resembling chorea, although Bernhardt is probably right in considering that they possess common characters.

[2] A condition known under various names—"spasmo-paralysis" (Marshall Hall), "spastic contracture," "hemiplegia spastica infantum" (Benedikt), &c.

[3] Art. "On Athetosis," 'New York Med. Record,' July, 1873, reproduced in the 'Med. Press and Circular,' Aug., 1873.

ceased during sleep, and much weight, therefore, cannot be attached to this distinction.

The examples I am about to adduce show, I think, how little reliance can be placed on spontaneity of movement as distinguishing the condition from post-hemiplegic disorders. Dr. Hammond's theory of the pathology of the disease is that it depends on some process of sclerosis, probably inflammatory, in some part of the central cerebral ganglia, corpus striatum, or optic thalamus. If so it will surely be a question merely of position and extent of lesion whether motor weakness does or does not accompany or precede the spasm. Hemiplegic numbness, present in some of his cases, points in the same direction. No case appears to have been carefully examined early in its history.

I have troubled the Society with these observations because, in the first I have to narrate, a condition of athetosis, characteristically similar to that in Dr. Hammond's cases, succeeded a distinct attack of hemiplegia. (Case 1 appended.) The patient was a young man, aged 23 at the time of the attack. The onset of the hemiplegia was sudden and without loss of consciousness; the paralysis was slight in degree, never preventing him from walking. It gradually passed, and in the limbs, which had been weak, at the end of two months, there came on slow irregular movements, which continued unabated when he was seen a year and a half after the onset of the hemiplegia. At that time his hand was never still for more than a few seconds. The fingers and thumb were in continuous slow movement,

acter, but ceasing during sleep.
low, irregular, flexion and exten-
in different fingers at different
hand in very strange attitudes.
associated with some lateral
In flexion the fingers became
st the palm, and until the spasm
mpossible for him to open the
nd. He had evidently a good

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deal of voluntary power over the hand and in certain
positions could modify the movements, but he could not
arrest them.   Extreme extension and extreme flexion were
the only positions in which the hand could be kept still
long enough to be photographed (see Plate XII, figs. 1
and 2); but it was usually constantly and slowly moving
in intermediate positions.   When firmly flexed the top of
the thumb commonly projected between the middle and
ring fingers.[1]   The foot was inverted, the toes in continual
but varying extension, the great toe especially, and the
short extensor was always in a state of conspicuous con-
traction.   The inversion of the foot was increased if he
stood, and occasioned him some pain.   There was no loss
of sensibility in the limb, no muscular atrophy or hyper-
trophy.   This condition persisted unchanged for twelve
months more, in spite of iodide and bromide of potassium
and a sea voyage.   He was then treated with the con-
stant galvanic current, no medicine being given, and the
hand gradually became steadier until in three months'
time all spontaneous movement had ceased.   The leg was
also galvanised, but with only slight diminution in the
degree of spasm.

In this case, then, we have a motor state identical, in
essential characters, with that described as athetosis, but
following a distinct attack of hemiplegia. There was nothing
to indicate exactly the position or the character of the ence-
phalic disease.   He had constitutional syphilis, and it is pro-
bable that a vessel, narrowed by syphilitic growth, became
occluded, leading to degeneration of tissue.

In the second case (Case 2 appended) slowly changing
spasm in the arm had succeeded two years' hemiplegic
numbness.   There was distinct motor weakness.   The diffi-
culty in moving the hand came on suddenly.   There were
slight spontaneous movements in the fingers when the limb

when the fingers became slowly flexed, abducted and, adducted, quite independently of her will. Voluntary movements were of course interfered with by slow in-co-ordination.

In each of Dr. Hammond's cases the motor disturbance has been preceded by sudden cerebral symptoms (convulsion, loss of consciousness), and in some was accompanied by hemiplegic numbness. There is thus evidence that the pathological change causing the disordered movement was, in some cases, secondary to a sudden lesion. Whether such lesion led to motor loss of power would be, as just stated, very much a question of position and extent.

I think, therefore, that, taking the facts of Dr. Hammond's cases as they stand, the initial hemiplegia in the case I have described affords no reason for separating it from the cases to which Dr. Hammond has given the name of athetosis.

If so we may approach with clearer light the question, do these cases differ essentially from the more familiar forms of mobile spasm in partially paralysed limbs ? In these the spasm succeeds a paralysis which at first is the chief symptom in the case. The spasm has a greater tendency to fix the limbs in certain positions, and if that position is altered it is regained with greater rapidity. There is in fact a fixed spasm superadded to the simple mobile spasm. It is this which constitutes the essential difference between these cases and those to which the name of " athetosis " has been given.

Several examples of this form are appended. Of these Case 3 (Plate XII, figs. 7, 8, and 9), is one of the most remarkable. The condition has succeeded an infantile hemiplegia. The limbs are shorter than the healthy limbs, but the muscles of the arm are so much hypertrophied by their constant overaction, that the circumference of the forearm is greater than that of the healthy arm. All muscles of the arm are in a state of spasmodic contraction, partly tonic and fixed, partly mobile and varying. The effect of this spasm is to hold the limb in

a certain position, in which it undergoes movements. The wrist and fingers are flexed; the latter by action of the interossei, slowly move, independently of her will. She has considerable voluntary power over the limb, especially when from time to time the spasm relaxes. The muscles of the face, especially the occipito-frontalis and the zygomatici, overact in certain movements. In Case 4 (Plate XII, figs. 4, 5, and 6), the symptoms have succeeded an attack of hemiplegia in early adult life. There is much more tonic spasm and a little unchanging rigidity, as well as slow mobile spasm, and both affect, as in most of these cases, the interossei in a very conspicuous degree, leading to the extension and even over-extension of the middle and distal phalangeal joints, while the metacarpo-phalangeal joint is flexed. The figures represent the habitual closed position of the hand and the attitude assumed in attempts to open it.

In another class of cases of post-hemiplegic disorder of movement the spasm instead of being slow and deliberate is quick and often jerky. In those already described the spasm gradually diminishes, without ceasing, in certain muscles, as it increases in others, and hence is produced the slow change of position. In this class, however, the spasm is more sudden, and ceases or almost ceases in one when it is developed in another, and hence quick movements result. These may be regular or irregular. The regular spasm causes certain alternating movements seen in tremor fine and coarse, and in the alternating movements of pronation and supination,[1] flexion and extension, abduction and adduction of wrist or fingers. The regular spasm may be continuous as in Case 18, or may occur only on movement as in Case 17. On the other hand the spasm may be irregular, producing jerking movements, rarely continuous, more frequently occurring only on voluntary motion, and thus causing wild jerking ataxy. In the interesting case brought before the Society by Dr. Hughlings Jackson early in the session, the clonic spasm

[1] As in a case mentioned by Dr. Bastian, 'Common Forms of Paralysis from Brain Disease,' p. 157.

was continuous and violent. In some cases the irregular spasm is slighter, producing choreoid movements. Of such continuous clonic spasm Case 12 is a remarkable example, while Cases 11, 13, 14, 15, and 16, presented such irregular choreoid spasm on movement only.[1] The two forms of spasm are often conjoined; in Case 10 slow, mobile spasm existed when the limbs were at rest, and quick jerking spasm on movement. In case 11 occasional clonic jerks occurred in muscles which were the seat of continuous slowly-changing spasm and (varying) rigidity; while in Case 12, in which the limbs were the seat of irregular clonic spasm, there was sufficient tonic spasm to maintain the limbs in certain positions—spasm which also ceased from time to time. When this irregular spasm is absent while the limb is at rest, and is developed only on voluntary movement, the effect is to produce mere inco-ordination, which may be slight and choreoid, or violent and jerking, and in the latter case may, as Charcot points out, resemble closely the movements of insular sclerosis.

All these forms of disordered movement possess certain common characters. They are far more frequent in the arm than in the leg, and when they exist in both (as in Case 11) they are more severe in the arm. The leg is usually very

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[1] These cases have been described, first by Weir Mitchell ('Amer. Journ. Med. Sciences,' 1874, p. 352), and after him by Charcot ('Prog. Méd.,' 1875, Nos. 4 and 6), as "post-hemiplegic chorea." Under this name, however, these writers include some forms of *slow* spasmodic movement, continuous, or on voluntary movement only, apparently such as in the case already spoken of. These cases of slow inco-ordination seem to me to bear little resemblance to ordinary chorea. I have preferred not to use the term "post-hemiplegic chorea" in speaking of any of these cases for two reasons :—First, I think the term tends to confuse together several easily distinguishable varieties of these movements, which may ultimately be found to depend on pathological processes of different kinds or in different localities; secondly, the word chorea has a long understood meaning as the name of a disease of certain course and relations, and were better not transferred to a symptom which can as conveniently, and more accurately, be spoken of as choreoid, if a longer descriptive designation is avoided. When this paper was written I had not seen Weir Mitchell's very interesting article. I have been glad, in revising this, to refer to those points in which it repeats Weir Mitchell's observations.

slightly affected; of the eighteen cases appended, Cases
6 and 11 are the only ones in which walking was seriously
interfered with. In the rare cares in which the leg is
affected, the paralysis of the arm is usually absolute, as in
Case 6, in which the leg possessed some power and was
the seat of variable mobile spasm and the arm was
absolutely paralysed, and the seat only of late rigidity.

The spasm may when slight, be confined to the hand;
when it involves all parts of the arm, the hand is always
the most severely affected.[1]   In the hand, the interossei,
as already remarked, suffer especially. It is remarkable,
too, that when the spasm occurs only on movement, it is
always greatest in, and may be confined to, movements in
which the interossei take part. In Case 9 this was
curiously noticeable. In Case 19 regular movements,
continuous, were due to the contractions in the two outer
sets of interossei.

The slow irregular spasm which occurs on movement is
clearly due in part to a diversion of the motor impulse along
an unintended path, or its irradiation over a wider region
than that to which it should have been confined.[2] In Case
6, for instance, a voluntary effort to move the foot only
resulted in its spasmodic inversion. In Case 3, an
attempt to extend the fingers resulted, first of all, in their
flexion apparently by an irradiation of the impulse of a
preliminary fixation of the wrist. In Case 8 the elbow
joint could not be moved without the occurrence of an
associated movement in the wrist and fingers.[3]

In some cases a still more curious phenomenon is seen,
a consentaneous movement of the limbs on the two sides.
In Case 9 for instance, whenever the healthy arm was
moved, the affected arm assumed a peculiar spasmodic
position, also assumed at the commencement of every

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[1] This is also remarked by Weir Mitchell.

[2] This has been termed by Nothnagel (' Arch. für Psychiatrie,' Bd. iii,
p. 214) " central irradiation of voluntary impulse."

[3] Hitzig has called attention to the spasm in associated muscles (mitbe-
wegungen) in " contracture" after hemiplegia. (Ibid., p. 314.)

convulsive seizure. I have seen the same bi-lateral association in one other similar case.[1]

Both slow mobile and quick clonic forms of spasm are related to voluntary power. They never occur in limbs the seat of an absolute paralysis. In some of the most marked cases the affected was as strong as the healthy limb. The voluntary power may be concealed by severe spontaneous spasm, mobile or clonic, but it always exists. Further,—in commencement the spasm coincides usually with, or follows quickly, the return of voluntary power; almost every case narrated illustrates this. In Case 4, however, in which the paralysis was for a long time absolute, slight spontaneous movements were noticed in the fingers some months before any return of voluntary power, but before the movements had become considerable some voluntary power had returned. In one remarkable case (Case 13) the commencement of the movements was associated by the patient with a violent effort to move the partly paralysed limb.

In some cases, as in Cases 2 and 13, the onset of the spasm has been described as sudden; more frequently it is gradual. In other cases a sudden change in its character is described. In Case 3 the arm had been for some years forcibly flexed at the elbow-joint, when one day suddenly it became extended and carried backwards, and so remained for months.

Whenever the spasm is continuous it is in most cases intensified by voluntary movement,—in some instances after a moment's cessation. Precisely the same form of spasm may exist in one case as continuous movement, in another on voluntary movement only. In several cases part of a limb, but vement. In Case 17 little fingers were in sm. On voluntary also the muscles of en in cases of "functional"

the thumb and first finger, and, though to a less extent, the muscles of the arm.

In certain conditions the spasm is increased or diminished. It is always slighter and often ceases when the limb is at rest: being increased by or produced by voluntary effort. It is increased by an effort to overcome it by passive force. It is lessened by warmth, increased by cold. It is less when the limb is rested, greater when it is fatigued. Lastly, it usually ceases almost entirely during sleep. In one or two cases slight movements continued during sleep, in others all movement ceased.

The movements, when slight, are sometimes increased by attention, just as are the movements in chorea. This was seen in Cases 9 and 13. It was more distinct in the cases of mobile and choreoid spasm in which the irregular combinations of muscular contractions are grouped in a more complex manner, than in the simpler regular alternating clonic spasm. The act of attention probably involves the transmission of a certain nerve influence to the muscles, and this may suffice to increase the irregular spasm.

Charcot has remarked that in post-hemiplegia inco-ordination, closure of the eyes does not increase the ataxy. Weir Mitchell observes that in some cases movement is interfered with by closing the eye, because the concentration of effort is interfered with. Charcot's rule is certainly not of universal application. In some cases the inco-ordination is certainly increased by closure of the eyes quite apart from concentration of effort. It was so in Case 16, in which the part was repeatedly tested, and the result was uniform. It may be related to the defective sensibility, as it is in locomotor ataxy.

No relation could be traced between the seat and form of spasm. Slow mobile spasm, quick, irregular choreoid spasm, regular alternating spasm, may be seen in either limb, but are certainly more common in the arm than in the leg. They affect the muscles of the hand and foot, or of the adjacent joints, more frequently than

the other parts of the the limbs. The quick spasm is especially uncommon in the leg.

The side affected seems to have little influence on the occurrence of the movements. Out of the eighteen cases, in eleven the movements were on the left, in seven on the right side.

The muscles in these cases are sometimes of normal bulk, sometimes actually hypertrophied (Case 3), sometimes wasted. Besides the variable tonic spasm already described, they are often the seat of a certain amount of permanent rigidity. This differs from ordinary late rigidity, especially in its seat, affecting the interossei rather than the long flexors of the fingers. The difference thus produced in the position of the hand is very striking. The irritability of the muscles does not present any uniform change, nor does that of the nerves. Many of the cases were tested with great care; in one or two a slight excess of faradic irritability was observed; in others no change.

Charcot lays stress on the association of hemi-anæsthesia with these movements. In ten of the cases appended the sensibility was carefully tested, and in four only could any defect be found. Nor was hemiopia frequent. The fields of vision were examined in many of the cases,[1] but in two only was any defect found (Cases 7 and 8), and in one of these the hemiopia did not correspond with the side affected with the hemiplegia and evidently was due to a separate lesion.

These conditions of motor disturbance are commonly seen as sequels of hemiplegia. But it is to be noted, and the fact is of especial interest in connection with the pathological position of athetosis, that any one of them may come on as a primary affection without preceding motor weakness. This is well illustrated by Case 11, in which a condition of limb, fixed and mobile spasm,

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[1] The field of vision was examined, I am sure, in several cases in which no note was made of it. For many years I have rarely examined a case of hemiplegia without searching for hemiopic defect.

resembling exactly that common in children after infantile hemiplegia, came on as a primary affection.[1]

What is the pathological condition which underlies these forms of motor disturbance ? The symptoms point clearly to damage to the grey matter of the brain, to local perverted nutrition of nerve cells, in consequence of which they overact, either spontaneously or on the stimulus of a volitional impulse which is by their overaction perverted or irregularly distributed.

In several of the cases appended there was other evidence of damage to grey matter. In some, unilateral convulsion occurred from time to time, involving the limbs which were the seat of the chronic motor disturbance (Cases 5, 7, 8, 9). In one (Case 9), the movement by which the fit commenced was identical with that constantly assumed by the limb in spontaneous spasm, and assumed also in a peculiar associated spasm which the limb presented when the healthy limb was moved. In Case 11 the spasm began in transient brief paroxysms, resembling slight convulsive seizures, which gradually became of longer duration, until they merged into a permanent condition. In the cases in which the motor disturbance succeeded hemiplegia, the conditions under which the symptoms arose, the suddenness of the onset, the wider area of the early symptoms, point to a lesion which damages more extensively than it destroys the brain tissue, and hence to softening rather than to hæmorrhage. The facts of the cases recorded suggest this view very strongly. Of the eighteen cases appended, in seventeen the onset of the symptoms was sudden. In four of these, the hemiplegia

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[1] The cases of ataxy in imbecile children, in which the limbs on both sides are in a state of mobile spasm with inco-ordination of movement, have been associated with athetosis by Dr. Claye Shaw, and may probably be ascribed to a similar pathological change. Many cases of " congenital chorea " are of this category. I have met with a well-marked instance in which there was no imbecility. Many of such cases are distinctly sequential to a bilateral weakness, and probably sometimes depend (as Weir Mitchell points out) upon injury to both sides of the brain received in birth.

came on in infancy with convulsions, and in three of them embolism or thrombosis was probable (onset during scarlatinal dropsy (Case 3), measles (Case 5), and after chorea (Case 8), respectively). In five cases the onset was during the day, and without any loss of consciousness (Cases 1, 2, 6, 13, 17). In two others (Cases 14 and 15) loss of consciousness occurred one or two hours after the onset of the hemiplegia. In one of these cases, post-mortem evidence showed clearly that there had been vascular obstruction and softening, and the resemblance between the two makes it very probable that the pathological condition was the same in each. Of the patients in whom there was loss of consciousness, one (Case 4) had suffered just before from acute rheumatism, and being a young man, was probably the subject of embolism. Thus out of the seventeen cases, there is in fourteen a probability that softening rather than haemorrhage was the initial morbid change.[1]

The greater frequency in the young of these forms of motor disturbance is in harmony with the readiness with which the growing nerve elements in them receive permanent damage, and suffer permanent alteration of function, from indirect shock or direct injury.

It seems essential for the existence of these disorders of movement, that the grey matter affected shall be in some connection, direct or indirect, with the volitional centre. At least as a matter of fact (as already stated), in no instance of post-hemiplegic spasmodic movement, was there an absence of voluntary power. It is of course obvious that continuity of the motor path from the volitional centre through the overacting grey matter is necessary for the existence of spasm occurring only on voluntary movement. It is less easy to understand why similar *spontaneous* spasmodic movement occurs in no cases except those in which there is such continuity of motor path. Possibly the association is indirect, the explana-

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[1] This conclusion is certainly borne out by a comparison of the details of other published cases, as, for instance, those of Weir Mitchell and Bernhardt.

tion being that these motor spasms occur only when the lesion causing the hemiplegia, &c. is in grey matter, and that they occur only when there is a large tract of grey matter undestroyed—damaged only—and that such imperfect destruction, necessary for the development of the spasm, permits also voluntary action. If this were the case it is evidently possible that choreoid movements might occur in a limb the seat of absolute paralysis as the result of a lesion outside the central ganglia, damaging, not destroying them, but isolating them from the cortex. This speculation is confirmed by a case related by Dr. Bernhardt,[1] in which such movements existed for a fortnight in an arm absolutely devoid of motor power, the paralysis being due to a very extensive lesion in the hemisphere outside the central ganglia.

In all the examples of slow mobile, and quick irregular clonic spasm, the hemiplegia in its general features was such as results from damage to the central ganglia of the brain.[2] In two cases only, of regular intermitting spasm, the symptoms pointed to a lesion lower down. The conclusion indicated by the symptoms is that in the forms of *irregular* spasm, spontaneous or on movement, the seat of the morbid action is the grey matter of the central ganglia, optic thalamus, and corpus striatum. This is in harmony with the speculation of Hammond as to the cause of " athetosis."

In one case (17) in which the spasm was peculiar in character the crossed face-and-limb paralysis points to damage to the pons. In the case of regular clonic spasm mentioned by Dr. Bastian the symptoms pointed to a lesion of the crus. It is possible that sometimes

[1] 'Berl. Klin. Wochenschrift,' 1875, No. 35.

[2] This is also the conclusion of Dr. Hughlings Jackson, who says, "There are several *mobile* counterparts of hemiplegia. There is hemichorea, there are certain cases of hemispasm, and there is what I call hemi-contracture, a mixture of palsy and spasm. I call these one-sided mobile symptoms 'hemi-kineses.' I believe that each of them depends on disease of the same *internal* region as does hemiplegia—the region of the corpus striatum; for the same external region is affected in each." 'West Riding Asylum Reports,' 1873.

damage to the grey matter of the convolutions may give rise to these symptoms. Eulenberg[1] and others have speculated that this is the common seat of the morbid process. No case appended suggests this, except Case 7, in which hemiplegia came on some months after a fracture near the vertex. But the occurrence of sight troubles points strongly to a second lesion more deeply placed.

What evidence is afforded by pathological anatomy ?

M. Charcot has found, in three cases of post-hemiplegic irregular choreoid spasm occurring on movement only, lesions involving in each case the posterior extremity of the optic thalamus, the most posterior part of the caudate nucleus, and the most posterior part of the corona radiata. He assumes that the symptoms depend upon damage to the latter, and that in these posterior fibres are some fibres endowed with special motor properties, the alteration of which gives rise to the choreoid spasm.

I have obtained an autopsy in one case only, the details of which possess considerable interest (Case 15). It is a case of post-hemiplegic incoördination, affecting the arm only, without spontaneous mobile spasm. The preceding hemiplegia came on somewhat deliberately in the course of an hour, was never quite absolute, and soon lessened. The ataxy developed with recovery of power, and was at one time so violent that if the patient suddenly raised an object from the table the arm would fly up over his head. The incoördination lessened before his death, which occurred about three years after the onset. The brain revealed but one lesion : a cicatricial induration of the optic thalamus extending across its centre beneath the upper surface, and approaching at its outer part, but not involving the ascending white fibres from the crus. No secondary degeneration could be found in the cord. The posterior columns were healthy. The microscopical characters of the lesion of the brain left little doubt that it was the

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[1] 'Handbuch der Krankh. des Nervensystem,' Leipzig, 1875 (quoted by Bernhardt, loc. cit.).

remains of an old focus of softening from vascular obstruction.

In this case then the lesion involved only the middle region of the optic thalamus; the nucleus caudatus and the fibres of the corona radiata were not damaged.

It may be difficult to understand, according to our received notions of the functions of the optic thalamus, how a lesion in that part can cause incoördination. Some explanation may be found in the observations of Crichton Browne on the abolition of reflex action in destruction of the optic thalamus[1], and the speculation of Meynert[2] and himself that the optic thalamus is a high reflex centre. Such a function involves efferent motor as well as afferent sensory fibres, and involves motor processes, no doubt in definite relation to the will, seated in grey matter, the ove-action of which may be concerned in the production of the symptoms in these cases.

But in two post-mortem examinations recorded by Weir Mitchell the seat of the lesion is said to have been, in each case, the corpus striatum. With this may be associated the fact that, in the cases which I have appended, hemianæsthesia did not exist in even a majority of the cases in which the sensibility was carefully tested. There is thus nothing in the history of these to point to a lesion of the thalamus. These facts suggest the probability that a similar change in the corpus striatum may give rise to similar symptoms.

The case I have recorded also goes far to negative the theory which has been put forward by one or two writers that these post-hemiplegic disorders of movement are due to secondary spinal changes. Here, at any rate, an extreme disorder of movement existed without spinal degeneration. The converse is also true of many cases, although of course of much less significance. Secondary degenerations of the cord are constantly met with in cases in which there is no mobile spasm or incoördination,

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[1] 'Stricker's Handbuch,' p. 752.
[2] 'West Riding Asylum Reports,' 1875.

even though the motor paralysis is not absolute. Further, the time of the development of these disorders of movement does not correspond to that at which the spinal degeneration is developed. The latter occurs at a certain, tolerably uniform, period after the cerebral lesion. The former may commence at a very variable date. In some cases it begins very shortly after the onset of the hemiplegia, in others not till long subsequently—not until long after the spinal degeneration, if present, must have been well developed. In Case 6, months elapsed before the return of voluntary power or the occurrence of spontaneous spasm. The sudden onset of the disorder of movement in some instances (as in Cases 2 and 13), is also hardly compatible with the idea that it is due to so gradual a cause as the spinal degeneration.

The distribution of post-hemiplegic spasm is of no significance, as to the spinal or cerebral position of its cause; but it has considerable significance in the cases in which a similar disorder of movement comes on without preceding hemiplegia. Then, the unilateral distribution of the spasm points very strongly to a cerebral cause.

The same conclusion is indicated by the fact, which I think the cases appended establish, that the character of the encephalic lesion influences the subsequent occurrence of these disorders of movement. It seems to be a more frequent consequence of softening than of hæmorrhage. But spinal degeneration is, as far as we know, related only to position and extent of destruction of nerve-tissue, not to form of damage. The influence of the latter on the occurrence of the movements indicates their direct dependence on the encephalic change.

In short, it seems probable that the forms of irregular disordered movement above described are due to lesions which damage rather than destroy the grey matter of the optic thalamus and corpus striatum, while there is reason to believe that some regular alternating movements may be due to a similar lesion affecting the grey matter in the pons Varolii.

Charcot, it may be mentioned, believes that the tremor sometimes seen in partly paralysed limbs is due to the secondary sclerosis of the lateral columns of the cord.

The condition of late rigidity was ascribed by Todd[1] to such an encephalic lesion as those which are here supposed to be the cause of mobile spasm, i.e. the changed function of cerebral tissue damaged by a lesion. Since the secondary degeneration of the spinal cord has been recognised it has become customary (following Bouchard) to refer this late rigidity to its influence. The chief reasons for believing that it is due to the spinal degeneration are, first, the association of the two conditions in time; late rigidity usually comes on about the time at which the degeneration has become developed. Secondly, the established fact that primary spinal degeneration may lead to rigidity of limb.

But when the phenomena of late rigidity are closely studied the condition is seen to possess certain features in common with the mobile spasm already described. It is far from uniform; it is slighter when the limb is warm, greater when the limb is cold. It can be lessened by gently rubbing the muscles, or by placing the limb in warm water. During sleep a large part of it passes away; the flexed fingers are supple and can be extended almost completely.[2] Whenever it is slight a voluntary effort to move the limb will always excite a more intense degree. If, for instance, the shoulder-joint possesses some power, and is moved, the spasm in the hand is increased. In the rare cases in which mobile spasm exists in the leg, and the arm is completely paralysed, this form of rigidity comes on in the arm at the same time that the mobile

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[1] 'Clinical Lectures,' Lect. xlv. Duchenne adopted a similar explanation, but assumed a chronic inflammation, of which there is, as Hitzig points out, very little evidence.

[2] This has been noted also by Benedikt ('Electrotherapie,' p. 219) and Hitzig (loc. cit.). The latter seems to regard it as a symptom of a special form of " contracture." I have found it a universal character.

spasm comes on in the leg, and the two may be increased and lessened by similar measures. This was very distinctly seen in Case 6.

Late rigidity only varies thus in part; there remains a certain amount of rigidity which does not change, and is probably due to actual alteration in the muscles. In most cases this constitutes but a small part of the total amount of "late rigidity." The varying element presents so many analogies to the mobile spasm that it is probably due to the same cause,—is the consequence, as Todd taught, of the irritative over-action of the damaged cerebral substance. We know that the irritation of the nerve elements involved in the cerebral lesion will cause hemiplegic rigidity, for it is seen in "early rigidity." The effect of reflex influences on lessening the spasm is no evidence of its spinal origin, since they may act in a manner similar to that in which a peripheral impression may arrest a commencing convulsion.

Two points of difference between ordinary "late rigidity" and that which accompanies and is part of the condition of mobile spasm, deserve mention. In late rigidity the long flexors of the digits suffer chiefly, the interossei scarcely at all. The fingers are extended at the metacarpo-phalangeal, flexed at the middle joints. In mobile spasm the affection of the interossei, and the position thereby impressed upon the hand, is always conspicuous. Again "late rigidity," as a rule, affects muscles which are completely removed from the volitional centre; voluntary power is absent, or almost absent. In

obscuring its leading feature, by which its position may be assigned to it. When spasm is described as "continuous" it exists during as well as apart from voluntary movement and produces, on movement, the special incoördination, which in other cases is all that is present. The "spastic contracture" as it is sometimes termed, especially frequent in early life, is composed mainly of varying tonic spasm, and mobile spasm on movement, but spontaneous mobile spasm, (*i.e.* continuous, apart from movement), is frequently conjoined, so that the bracket in the table might have been extended so as to include "athetosis" as a factor in this condition. Varying tonic spasm may sometimes, as in Case 12, be associated with the irregular jerking spasm of the first group. The associated bilateral spasm is not included in the table.

It may be again remarked that although these forms of disordered movement are termed post-hemiplegic, most, perhaps all of them, may come on as primary affections, without preceding hemiplegia.

The prognosis in post-hemiplegic disorders of movement is grave, but I do not think it is so hopeless as is sometimes asserted. The ataxy frequently ceases, as in the case I have alluded to. Mobile spasm developed in early life may lessen in later life, but rarely disappears. The athetotic form has hitherto persisted in every case which has been described, and I believe that the case I have brought before the Society to-night is the first in which, under treatment, the spontaneous movements have entirely ceased.

In this case the recovery appeared distinctly due to the use of the constant current. No other remedy was employed at the time. Other remedies, bromide, &c., had been given without benefit. The patient was, so far as food, &c., were concerned, in the same circumstances as he had been in before. His foot, however, was not benefited in the same degree, and still continues inverted. In some other cases in which I have tried the same remedy marked improvement has taken place. In Case 8 the result was

Post-hemiplegic disorders of movement.

Quick, clonic spasm, of intermitting type
- Regular (continuous, or on movement)
  - Tremor { Fine / Coarse
  - Certain, regular movements, due to interossei pronators, &c.
- Irregular (continuous, or on movement)
  - Choreoid } Continuous spasm, or incoördination of movement.
  - Jerking

Slow, mobile spasm, of remitting type
- Continuous = "Athetosis."
- On movement = Slow, cramp-like incoördination } "Spastic contracture" of hemiplegic children.

Tonic spasm, varying .
Fixed rigidity, unvarying .
- Of interossei, conspicuous
- Of flexor longus digitorum, conspicuous = late rigidity.

to restore a large amount of use to a limb from which it had been long absent. In a case of ataxy not included among these it so steadied the patient's hand (left) as to enable him, a draughtsman, to keep the paper steady on which he was drawing, and so to earn his living. In the case of congenital and bilateral spasm alluded to in the note on p. 282 the improvement, although slight, was distinct, as the patient became able first to stand and then to feed herself. The remedies which have seemed to give most relief next to galvanism have been bromide of potassium and Indian hemp.

CASES.

The following series of cases are some which have come under my observation, most of them at the Hospital for Paralysis and Epilepsy. Some of them were under the care of Dr. Ramskill, Dr. Radcliffe, Dr. Hughlings Jackson, and Dr. Elam, who kindly permit me to use my notes of the cases. Case 12 came under my observation at University College Hospital, and for permission to publish it I have to thank Dr. Reynolds. The other patients were under my own care at the Hospital for Paralysis, with the exception of one or two who were seen in private.

CASE 1.—*Slow, continuous, mobile spasm (athetosis), in right arm and leg, supervening on a slight attack of hemiplegia. Improvement under treatment.*—P. P—, a healthy looking man, aged 24, presented himself in October, 1874, with certain disordered movements of the right hand and right foot. Its characteristic was slowly changing spasm, causing a distorted position of the fingers and toes and interfering greatly with voluntary movements.

His history was that at eighteen years of age he contracted syphilis, a hard chancre, which was followed by sores on the forehead. At 23, a year before he came under observation, he married, and four months afterwards, on rising one morning, he felt sick but did not vomit. He walked to

the watercloset, then back to bed, and found, on returning, that his left side was weak, his foot dragged. He seemed to his wife quite in his usual mental state. He slept for two or three hours, and on waking found his left hand weak, so that he could not dress himself, and a little " numb." His face was noticed to be unequal. He walked into another room, dragging his leg. He had no pain in the head. Next day he walked half a mile to church, and after church went for a walk, shuffling his foot. During the next few days the weakness steadily decreased, and at the end of a week he was able to return to his employment as clerk. For six weeks he walked with a slight limp, but at the end of that time all the symptoms of weakness disappeared. About two months after the onset of the hemiplegia he found his foot painful at the instep when he walked, and this pain continued. About four months after the onset he found the movements of his hand gradually become unsteady, and irregular spasm slowly developed in it. Except for this, and for slight attacks of giddiness, the patient believed himself to be quite well, but his wife observed distinct failure of memory and a clipping of words, not present before the attack.

When he was seen, eight months after the attack and four after commencement of the spasm in the arm, it was difficult to say, on account of the spontaneous spasm, whether any hemiplegic loss of power remained. He had certainly considerable strength in the hand, and could walk many miles, with a limp due to the position of the foot. There was no inequality of the face or deviation of the tongue. The most common position of the hand was with the fingers flexed and the thumb within the first two fingers, its tip projecting between the middle and ring fingers. The hand was then almost still, but he was conscious of a firm pressure of the fingers against the palm, and of the thumb against the fingers (Plate XII, fig. 1). When the hand was in this position he could not voluntarily extend it, often he could not extend the fingers with his left hand. After a few minutes the

spasm relaxed, so that with his other hand he was able to extend the fingers. The fingers then slowly became strongly extended and separated, the thumb being also extended (fig. 2), the position of the fingers varying. From time to time, the hand if closed would slowly open out in extension entirely apart from his own will. The complete extension was usually only maintained for a few moments, then one or more of the fingers would become bent at the metacarpo-phalangeal joints, remaining extended at the others, and then first one finger and then another would slowly become entirely flexed until the hand was closed as before. The spasm which kept it closed was always varying, and an attempt to open it seemed at first to increase the force of closure. At first, perhaps, when the hand was extended, the forefinger would first be abducted, then the little finger, then the two first fingers would be brought together and afterwards slowly flexed, and lastly all the fingers flexed. He could keep the hand closed or open for a few moments by a voluntary effort, when already in that position; he could expedite, by his will, extension, or flexion, when it was occurring, but had little other power over the spasm. As a rule the hand was in some intermediate, constantly changing position between flexion and extension. The spasm was attended by some pain in the muscles of the forearm, relieved considerably by pressure upon them. There was no movement in the muscles of the elbow or shoulder, over which he seemed to possess full voluntary power; but slight spasm was afterwards noticed in the biceps. There was no wasting or hypertrophy of the muscles, the maximum circumference of the right forearm below the elbow being 9⅝ inches and that of the left 9¼.

The foot was habitually inverted, the tibialis anticus constantly overacting, and the toes were over-extended, the short extensor of the toes contracting strongly and standing up conspicuously in the back of the foot (see figure 3). This condition was more fixed in the foot than in the hand, but changed from time to time, the foot

becoming more or less inverted, the toes more or less extended, especially the great toe, which was over-extended at both its joints. The other toes became extended at the metatarso-phalangeal joint, and flexed at the other joints, especially the smaller toes. Circumference of left calf ⅛ inch less than the other. The muscles of the left limbs were a little less irritable to electricity than those of the right, both to faradisation and the constant current, the difference existing both in nerve and muscle.

Sensation in the limbs was perfectly natural.

The patient was seen only twice at this time, as he was leaving for a sea voyage. He was ordered large doses of iodide and bromide of potassium, which he took regularly for some months. In a year's time he returned to England and was then taken into the hospital. His condition was unchanged, the hand was in the same constant involuntary movement of irregular extension and flexion. He could direct the movements of the hand a little better, but was quite unable to pick up a small object. He was still conscious of no difficulty about the upper part of the arm, but on close examination it was found that extension of the elbow produced some pain in the biceps, and he remarked that he constantly found the elbow becoming strongly flexed and the hand pressed against the chest. No medicine was given to him, his food, &c., was nearly that he was accustomed to out of the hospital. The continuous galvanic current was applied daily, the positive pole being placed on the neck, and the negative pole on the overacting muscles and on the hand and foot. After each application he thought that his hand was steadier, and in a month the spontaneous spasm was considerably less. He could keep his hand flexed or extended, and could manage, although with difficulty, to pick up even a very small object. His great toe was less extended, and his walk less halting. The applications of electricity were continued during the next two months, and the spontaneous movements became slighter and slighter, and finally ceased altogether. A little stiffness in the movements of the

fingers was all that remained. In his leg the improvement was slighter. The foot remained inverted and the toes extended, but the pain in the ankle was considerably relieved by bromide of potassium with Indian hemp.

His general health remained good, but nearly two years after the onset of the hemiplegia two nodes appeared in his skull, and rapidly yielded to iodide of potassium.

CASE 2.—*Slow mobile spasm in left hand, in part spontaneous, but chiefly in certain positions ; onset sudden. Preceding defect of sensibility. Slight motor hemiplegia.*— A. E. W—, a widow, aged 38, came under my care in March, 1876. She had never been pregnaut, and there was no syphilitic history. Three years previously she became liable to attacks of intense pain in the head, general, with violent sickness, the pain being worst at night, and so severe as to make her roll on the floor. Soon after the commencement of the attacks of pain, objects at a distance appeared to her to be double. After a few months this double vision ceased. The attacks of headache continued for a year or more. Two years before being seen she found that the left leg and arm were " numb," the sensation being as if the limbs were " asleep." At the same time her leg was weak. The numbness, and weakness in the leg continued. A fortnight before admission she suddenly found that she could not use the right hand so well as before, could not button her boots with it although she could move it freely. At the same time her leg seemed weaker.

When first seen her state was nearly as follows :— Mind clear and intelligent. No defect of speech. The sensibility to touch in the right arm and leg were distinctly defective ; a slight touch was not felt, a firmer touch was felt, but she uniformly asserted that it was much less distinct than on the other side. There was no defective or altered movement on the face or tongue. The right arm was weaker than the left (dynamometer, right 10 ko., the left 18 ko.). The fingers could be moved pretty

freely. When the hand was at rest the fingers were all slightly flexed at all points, but they were not quite still; slight slow movements of flexion and extension, and inter-osseal abduction and adduction could be observed. The movements were not conspicuous, but were distinct on close observation. When she attempted to extend her fingers they became widely separated, some being extended at once, and others, especially the middle finger, remaining for a time flexed at the metacarpo-phalangeal joint and extended at the others. After a time all were straightened (see Plate XII, fig. 10). If the hand was held in a position of extension of the wrist, the slow irregular movements were more marked, and were chiefly movements of abduction and adduction, and of flexion of one or two of the fingers at the metacarpo-pha-langeal joints, the little finger especially being frequently slowly separated from the others and brought to them again. These movements were quite involuntary and she had no power to prevent them. This spasm produced an incoördination which prevented the execution of any delicate voluntary movement. She could not unbutton her dress, not from want of power, but from the superadded cramp interfering with the muscular action, so that the thumb slipped off her forefinger when she attempted to hold any object between the two.

There was no defect in the field of vision, no ophthal-moscopic change.

The leg was a little weaker than the other, but there was no inversion of the foot or defective coördination.

During the six weeks she was under observation there was considerable diminution in the spontaneous movements, but the mobile spasm on voluntary movement continued, although slight. In other respects her state was unchanged.

CASE 3.—*Mobile and fixed spasm of left arm with muscular hypertrophy. Over-action of facial muscles. Sequel of infantile hemiplegia.*—M. H—, female, æt. 29. At six

years of age, while lying ill with scarlatinal dropsy, left
hemiplegia came on with a series of convulsions. Fuller
details of onset could not be obtained. The paralysis
appeared to have been complete at first, and recovery of
power to have been slow. The spasm in the arm pro-
bably came on slowly about a year after the onset of the
hemiplegia. The arm was then bent at the elbow, the
forearm flexed against the arm so forcibly that it
could not be straightened, the fingers and wrist being
flexed. This posture was maintained for some years, and
it is affirmed by the patient and her mother, with circum-
stantial detail, that the posture of the arm suddenly
changed : instead of being flexed it became extended, the
elbow straight (but the fingers still flexed), and the whole
arm carried backwards behind the trunk. It continued
in that position until the age of twenty-five or twenty-six,
when she became able to flex and extend the elbow at
will, the spasm in the meantime persisting.

State on Examination, June, 1875.—The left arm is
shorter than the right, the left ulna measuring nine inches
in length while the right measures ten inches. Neverthe-
less the left arm is considerably the thicker ; the maxi-
mum circumference below the elbow is in the left nine
and a half, in the right nine and a quarter inches ; the in-
creased size depends on muscular hypertrophy (Plate XII,
fig. 7). The muscles are in a state of continuous but
varying spasm. The common position of the arm is by
the side with the elbow extended, the wrist flexed, and
the fingers flexed, but in a varying manner, the ring and
middle finger being flexed at the metacarpo-phalangeal
and also at the middle joints; while the forefinger and
little fingers are flexed only at the former, being extended
at the two phalangeal joints (interosseal position). The
thumb is over-extended at all joints. The position of
the fingers changes, and the thumb is often pressed with
great force against the fingers, so as to press back the
last phalanx. At other times the first finger is extended
at all joints, the others flexed at the metacarpo-phalangeal

joints (Plate XII, figs. 8 and 9). The fingers are occasionally over-extended at the middle joints. It is with difficulty that the fingers can be held still to be photographed. She has little power of arresting their movements, which go on slowly but continually.

When the hand is at rest for a few minutes the spasm becomes much slighter, and she can then, with the other hand, extend completely the phalanges, and almost extend the wrist. A voluntary effort brings back the spasm in all its uncontrollable intensity. When she is asleep the hand is always still, and her mother thinks, quite limp. The arm during sleep is sometimes by her side, sometimes across her chest. Sometimes the wrist is flexed during sleep.

The leg is much stronger than the arm. It was at first "turned" as she walked upon it, but improved as she gained strength. She can now walk a fair distance, but the foot is always a little inverted and the toes turned up, and this condition is increased by any attempt to move the foot.

Her face presents marked inequality (fig. 7). At rest there is little difference, the left angle of the mouth being sometimes, but not always, a little higher than the other, and the transverse wrinkles on the forehead commonly more marked on the left side. Slight voluntary and emotional movements are much greater on the left side than on the right; this is especially seen in the action of the zygomatic and frontal muscles; the naso-labial furrow being deep, the eyebrow raised, and the forehead drawn into transverse wrinkles. Closure of the eyes and frowning are equal on the two sides. A stronger movement, as in laughing, is also equal or even a little more marked on the right side.

The electric irritability in the muscles of the limbs of the two sides is equal.

The condition of the patient remained unchanged while under observation.

CASE 4.—*Fixed and mobile spasm in muscles of left hand, especially in the interossei; slight in foot; sequel of hemiplegia.*—A. M—, a young man, at the age of 19 had an attack of acute rheumatism, which left, so far as he knows, no heart disease. No history of syphilis could be obtained. At twenty-three, left hemiplegia came on suddenly without premonition of any kind. The onset occurred during a short railway journey lasting twenty minutes. He had not hurried to the train. Consciousness was lost during part of the journey and regained at its close, but he did not know that he had become paralysed until informed by a surgeon to whose house he was carried. (His fellow travellers were all strangers to him.) He was able to give his name and address, and was taken home in a cab, after which he again lost consciousness and remembers nothing more for three weeks. Then he could speak well and could swallow fairly well. His face was unequal; his arm was powerless, except for slight motion at the shoulder-joint. He could move the leg a little at the hip-joint. His power of retaining urine was slightly impaired. He thinks there was no affection of sensation.

Power was regained more rapidly in the leg than in the arm. In three months he could walk round a room, and in a year could walk ten miles, but with a limp. Twelve months passed before there was the slightest power of moving the fingers. Then one day he noticed that one of his fingers moved without his will. The slight movement was repeated, and shortly afterwards another finger moved a little. This involuntary movement increased during the next few months, and about six months after he noticed the first spontaneous movement he became able to flex the fingers slightly by a voluntary effort. Both voluntary power and spasmodic movements gradually increased, and about two years after the onset a state was reached in which up to the present time (six years after the onset) no noticeable change has occurred.

He is now a healthy-looking man of twenty-nine. His heart presents no evidence of disease. The impulse and sounds are normal.

There is no trace of paralysis in face, tongue, or in any of the cranial nerves.

His left arm is a little thinner than the right (maximum circumference, right forearm, about three eighths inch more than left). Movement at the shoulder and elbow is free and strong. At the elbow there is some resistance to passive movement, from slight muscular rigidity, and he says the elbow has a tendency to become flexed. The "natural position" seems to be with the hand against the upper part of the chest in front. The forearm is habitually over-pronated, so that the palm of the hand looks outwards. He can supinate it, but not completely. He can flex or extend the wrist readily, and can flex the fingers well, grasping almost, though not quite so strongly as with the right hand. The fingers are always flexed at the metacarpo-phalangeal joints, sometimes flexed at the other joints, but very commonly extended at the second and third joints, so as to be held straight, and at a right. angle to the metacarpal bones. They are a little inclined to the ulnar side (Plate XII, fig. 4). The thumb is usually pressed firmly against the first finger, but with varying force. Slight spontaneous movements occur in which the thumb is occasionally moved outwards a little, and then pressed against the first finger. The little finger is flexed a little, and then extended slightly. Then, first one and afterwards another finger is slowly flexed. The interossei can be seen from time to time to contract slowly, especially the first (abductor indicis) which often contracts when it produces no movement of the (already flexed) forefinger. When the fingers are all flexed and he attempts to extend them by a voluntary effort, the first effect is an increased flexion, apparently part of a preliminary fixation of the wrist (irradiation of impulse). The fingers are then straightened at the two distal joints, and to a variable degree at the

metacarpo-phalangeal, the little and ring finger more than the others, and in the effort the fingers are separated both laterally and vertically. The thumb in the effort is sometimes extended, but often is flexed still more strongly, and getting over the forefinger prevents its extension (see figs. 5 and 6). Complete active extension is impossible. With considerable force the fingers and wrist can be passively extended, and if laid on a flat surface they will remain so, the spasm becoming much slighter. It does not, however, cease entirely, and as soon as the hand is raised, by himself or by another person, the fingers become flexed by the interossei, the hand assuming the characteristic position. This spasm interferes with his use of the hand, but there is no other incoördination of movement. He is able with it to hold paper steady on which he is writing. During sleep the hand is " very open," knuckles upwards, with a very slight movement in the thumb and little finger.

His left leg is a little thinner than the right, right calf 14½ inches, left 13¾ inches maximum circumference. He can move the hip and knee-joint well, but complains of a difficulty in getting the heel on to the ground, so that in walking the ball of the foot is pushed along. The toes are straight when the foot is on the ground; when it is raised, however, the foot becomes inverted, and the great toe over-extended, and the extensor tendons of the other toes are tightened. Presently, quite apart from his will, the toes undergo slight, slow, successive movements of flexion, most considerable in the great toe.

There is no uniform difference in the electric irritability of the muscles of the limbs of the two sides. Sensation in the left arm is a little affected. A touch is felt, but a little differently from the right; a pinch is felt rather *more* acutely, but he is able to distinguish differences in temperature much less readily than with the right hand.

During the three years in which he has been under occasional observation, the only change in his condition has been a slight diminution in the spasm of the inter-

ossei, so that the hand can be opened rather more readily, and kept open with slighter force.

The constant current has not in this patient made any appreciable difference in the movements.

CASE 5.—*Extreme fixed, but varying, spasm in left arm, after infantile hemiplegia, ceasing during sleep, lessened by warmth, &c., unilateral convulsion.* (Under the care of Dr. Radcliffe).—A. A—, a girl, æt. 15, had been hemiplegic since 2½ years. The paralysis was on the left side and came on suddenly, it was said with a fit, during an attack of measles. The leg was powerless for a time but slowly recovered power; the arm passed gradually, as it recovered, into the state of mobile spasm which it exhibited when the patient came under observation (June 1872), and the following note was made.

Sight and hearing unimpaired; the masseter and temporals unaffected. The lower part of face and the tongue are considerably weaker on left side, the latter deviating considerably on protrusion; the whole left side of the chest are smaller than the right and moved less on deep inspiration. The left arm is of the same size and as well nourished as the other. She possesses considerable power over the limb, but its use is interfered with continuous mobile spasm. The hand is usually strongly flexed on the forearm; the fingers flexed, the thumb bent in, and the forearm pronated, the pronation being carried to such an extreme that the palm is directed outwards as the arm hangs by her side. The elbow is usually extended and rigid. When the limb is in this position she cannot overcome the spasm by voluntary muscular contraction, and the spasm, varying a little from time to time, maintains its general features and degree. If she wishes to flex the elbow she first abducts the arm by the deltoid, and when it is away from her side she can flex the elbow, and then move her arm freely so as to touch the top of her head, &c. She cannot, in any posture, overcome the spasm in the fingers and wrist by simple muscular effort.

With her other hand she can open the closed fingers and extend the wrist, and then all the fingers can be extended without pain. As soon, however, as the hand is let go the deformed position is resumed.

During sleep the whole or almost the whole of this spasm ceases. It becomes much less in warm water, and she can then get the fingers and wrist extended by a little manœuvring. If she begins by attempting to extend the wrist she fails, but if she begins by relaxing the flexors to the utmost by flexing the wrist strongly, the fingers presently straighten, and then by a sudden movement she can get the wrist extended, the fingers also remaining straight. After a little time there is strong abduction of the hand to the ulnar side, and then it resumes its attitude of extreme flexion.

The leg is almost as strong as the other, no spasm or rigidity exists in any part of it. She can walk, with a slight limp, a long distance.

Since her seventh year she has been subject to what are termed fits, loss of consciousness with tonic spasm in the paralysed limbs, lasting a few minutes and preceded by an epileptic aura. Some of the attacks were witnessed; in slight ones there was not loss of consciousness.

During the time the patient remained under observation there was no change in her condition.

CASE 6.—" Late rigidity" in arm, mobile and fixed spasm in leg, after hemiplegia.—A widow lady, æt. 52, was seized with left hemiplegia while apparently in perfectly good health. The hemiplegia was for a time complete, but after some months slight power of movement returned in the leg. Two years after the onset, the face was slightly paralysed in its lower portions, the tongue did not deviate. The arm was still completely paralysed, and the seat of well-marked "late rigidity." The elbow could not be completely extended; the fingers were extended at the metacarpo-phalangeal joints, flexed at the middle and distal joints by the contraction of the long flexor muscles.

Ordinarily, they could not be extended except when the wrist was flexed. The amount of rigidity, however, varied very considerably at different times. When the hand was cold the flexion of the fingers was much greater and less amenable to passive extension. When the arm was warm the fingers could be extended much more by slow pressure. During sleep the flexion of the fingers was very slight; they were almost straight, even though the wrist was also extended. At all times if the rigidity was slight it could be at once intensified by an attempt to move the limb. There was moderate wasting of the limb.

The leg possessed considerable power, but its use was interfered with by slow mobile spasm. The foot was generally over-extended and inverted, the posture varying. By gentle pressure the foot could in a short time be brought into its normal position, but an effort to move the limb, and even a painful impression upon it reproduced the inversion; on attempting to stand it became extreme. There was slight permanent shortening of the overacting muscles, so that complete flexion of the ankle was impossible, but the amount of permanent rigidity bore but slight proportion to the amount of mobile spasm. A voluntary effort to move the foot seemed perverted before it reached the muscles, and it was difficult to ascertain how much power over the flexors of the foot she possessed; because an effort to flex or evert the foot caused only increased extension and inversion. There was no pronounced defect of faradic irritability in any of the muscles of the arm or leg. There was no defect of sensibility.

She was treated by the constant current to the overacting muscles, faradisation to their opponents, and some orthopædic mechanism. Some improvement occurred, but afterwards, the treatment being neglected, the muscles wasted, and the permanent rigidity and shortening of the overacting muscles increased.

CASE 7.—*Slow mobile spasm of hand, increased by attention, the result of a hemiplegia after brain injury.*

Unilateral convulsion.—Henry C. B—, æt. 12, several years ago suffered from depressed fracture of skull, the result of a fall, which was raised, three months after the fall, by Sir James Paget (at St. Bartholomew's Hospital). No unilateral symptoms were present before the removal of the bone. There is now (January 1876), a soft depression, in the upper part of the right side of the frontal bone, close to the coronal suture, about an inch in diameter. He returned home apparently well, when suddenly, a year after the accident, he had a fit which left paralysis of the left arm and leg, and blindness of the left eye. Since that time four or five fits occurred weekly until the commencement of the attendance at the hospital.

Present state.—In each fit consciousness is said to be lost, the convulsion is violent, but affects the left limbs only, and begins with a movement of the left hand up to the head. The left arm and leg are weak, the arm especially. He can move the limb freely but cannot compress Charrière's dynamometer. The whole of the left arm is much thinner than the right. When his attention is not directed to the left hand it is still and relaxed by his side. As soon as any attempt is made to move it, as soon even as his attention is fixed upon it, spasm comes on; the wrist is over-extended, the fingers are flexed at their metacarpo-phalangeal, extended at their other joints (interosseal position), the thumb is pressed forcibly against the first finger. Occasionally, even when in spasm, the distal joints of the fingers are flexed.

The leg is thinner than the other, although the wasting is less than in the arm. The muscles are weak, especially the extensors of the foot. There is no rigidity of the arm. There is no paralysis of the face or tongue. The left eye is almost blind, very little perception of light. Grey atrophy of the optic disc; vessels large; slight disturbance of choroid. Right eye, left half of field of vision lost; right half fairly complete.

CASE 8.—*Infantile hemiplegia (left) ; movements of hand,*

*after recovery, interfered with by spasm in associated mus-
cles ; improvement under treatment.*—A. M—, a healthy
looking girl, æt. 15, became hemiplegic at two years of
age, the paralysis being discovered at the end of about a
week of frequent left-sided convulsions. The onset of the
convulsions was sudden, the child having been, up to that
time, in apparently good health.

The paralysis was at first absolute ; after a time some
power gradually returned in the arm and leg, but the
slight movement of the hand became interfered with by
spasm, such as existed when she came under observation.
For several years she had been liable to slight convulsive
attacks involving the left side only.

When first seen on June, 1875, her state was as
follows :

There is no mental defect. No paralysis of any cranial
nerve. The left arm is a little smaller than the other, the
ulna being half an inch shorter, and the maximum cir-
cumference of the forearm half an inch less. She has
considerable power over the arm, can move the shoulder-
joint freely, and can flex and extend the elbow-joint,
though not so quickly as in the right arm. She can
move her hand and fingers, but so much spasm occurs in
associated muscles that she is scarcely able to use the
hand for any purpose. The grasp is firm, though not strong
enough to move Charrière's dynamometer. The grasp
cannot be relaxed readily, and the fingers, on extension,
are straight for a moment, but speedily become (in-
voluntarily) a little over-extended at the metacarpo-
phalangeal joints, and flexed at the other joints, while
the thumb is over-extended at each joint. By an effort
she can extend all the fingers, and they then separate
from one another and spread out. When she flexes or
extends the elbow-joint the wrist also is flexed or
extended ; the fingers separate widely. The wrist
cannot be moved without the fingers. The interossei can-
not be put into separate action ; the index finger can be
extended alone at the middle and distal joints, but

cannot be extended at the metacarpo-phalangeal joint except with the others. In almost any movement the little finger is abducted. When voluntary movement ceases, the hand is at rest, no spontaneous movement continuing.

The leg can be moved well and strongly at the hip, and knee, and ankle, but she cannot extend the toes or flex the ankle so much as in the other leg. There is no spasm on movement resembling that of the arm, but there is some excess of reflex action, the application of a moderate faradic current to one muscle producing contraction in all adjacent muscles.

The electric irritability of the limbs presents no uniform change. The extensors of the fingers are more, those of the thumb are less irritable in the affected than in the healthy arm. Sensation is normal.

The treatment adopted was the internal administration of bromides and nervine tonics, and externally the constant current to the arm; positive pole to the neck; negative to the muscles of the arm and hand, partly stable, partly labile; faradisation to the weaker muscles of the leg, extensors of the toes and tibialis anticus—all twice weekly.

In two months a marked improvement had taken place in the freedom of the movements of her hand. She could flex and extend the fingers separately, so as to play a " five-fingered exercise " on the piano, and to hold her needlework, or hold a paper which she was folding (neither of which she could do when the treatment was commenced). She was able to move the dynamometer up to 6 ko.

Two months later the power of the hand had increased still more, and she could move the dynamometer to 22 ko. The hand could be opened and closed with perfect steadiness, with the first three fingers together, only the little finger being separated from the others. She could extend completely the first two fingers together, but was still unable to extend completely the first finger alone.

However, she was able to use the hand continually in various ways. Her leg had improved, although less than the arm. She was able to raise the toes much better in walking.

CASE 9.—*Slow mobile spasm in left hand, chiefly on movement, succeeding chorea and unilateral convulsions. Associated movements when opposite hand is used, and a similar movement at the commencement of each .fit.*— Charles B—, æt. 11, was well till an attack of chorea at seven, which was ultimately, if not at first, confined to the left side. The first convulsion occurred at nine years of age, and was general. The fits recurred, and permanent weakness in the left arm and leg was noticed.

When he came under observation (in May, 1876), the following note was made :—There is more movement in the lower part of the face on the left side than on the right side (apparently from overaction of the former). The tongue is unaffected. The left arm is weak in all parts (dynamometer, right 20 ko., left 10 ko.), the left leg scarcely weak. Placed at rest, the left hand is nearly still, the only movement being a little slow continuous extension of the thumb, which goes on till the last phalanx is over-extended, and then ceases. Occasionally the fingers, apparently involuntarily, become all flexed at the middle joint. He can move the hand so as to hold an object, and pick up a small object without showing any incoördination. A complex movement such as unbuttoning his waistcoat is interfered with by some slow spasm in the muscles. The wrist joint always becomes strongly adducted and somewhat over-extended, the fingers are considerably over-extended, and spread out. When the fingers are placed together he can open and close the hand readily. Keeping the fingers together, but cannot separate the extended fingers without considerable difficulty, the fingers become over-extended, especially the outer ones, and for a long time the two first fingers remain in contact. When he uses the right hand, if he puts the left hand on his knee

and presses it firmly against the leg he can keep it there, the thumb becomes strongly flexed into the palm; the two first fingers are straight and in contact, the others become over-extended and separated. If he does not keep the hand in this position, whenever he uses the right hand, the left hand is raised, the elbow flexed so as to bring the hand up to the shoulder with the palm forwards. Sometimes the hand is held down, but then the wrist becomes over-pronated, so as to twist the arm until the palm looks forwards. The foot is a little inverted.

The convulsive attacks to which he is subject begin with a sensation to the left of the epigastrium. This passes up to the left shoulder and then down the left arm. The head is turned to the left, and the left arm is raised, the wrist extended, and the palm forwards (just as in the associated movement when the right hand is used). He retains consciousness until the hand is going up, and then becomes insensible. The convulsion is confined to the limbs on the left side. Sometimes a sudden start or shake as the hand is going up will arrest the fit.

Case 10.—*Slow mobile spasm of right arm (spontaneous), and rapid jerking spasm on movement; after hemiplegia.*—M. B—, a married woman, at the age of forty-nine became hemiplegic on the right side. After much nursing at night she suddenly felt " pins and needles " in her face and side, became unconscious for a short time, " an hour," and on recovery of consciousness her face was " drawn," and her right arm and leg powerless and insensitive. Power was gradually regained; in two months she was able to walk a little, and had recovered some movement in the arm, so that she could " shake hands." The arm was then quite steady. After three months the power of feeling in the arm and leg gradually returned, and about the same time the movements of the arm became unsteady.

When seen (in June, 1873), two years after the onset, the following note was made:—Face scarcely affected,

very slight weakness in the right side; tongue protruded straight. The left arm is perfectly strong and steady; the right arm possesses considerable power, but its use is interfered with by spasm. It is not wasted; the muscles are as well nourished as in the other arm. When the hand is at rest in her lap, there are slight slow " cramp-like " movements of flexion and extension of the fingers, which are quite independent of her will. On being told to extend the fingers this is done, but slowly and irregularly. At first one or two fingers are extended completely, and one or two others are extended at the two phalangeal articulations, but flexed at the metacarpo-phalangeal joint (from action of the interossei). Ultimately all the fingers and the wrist are completely extended. When, however, she raises her hand from her lap, the whole arm is moved irregularly, being rapidly jerked to and fro through a space of three to six inches. All the muscles, in all parts of the arm, seem to participate in this irregular spasm. When desired to take hold of a stethoscope standing on the table near her, the hand is thrown wildly about, and finally after several misses she makes a " dart " at the object and catches it. She can flex and extend the elbow much more steadily when the arm is against the side, than when it is abducted.

The leg is much stronger than the arm. She can walk one or two miles with a slight limp. Its nutrition is unimpaired. It appears quite steady, but when she lies down the great toe becomes a little over-extended. Sensation in both arms and leg is now quite good.

CASE 11.—*Variable tonic spasm in right arm, with occasional slight clonic spasm; rigidity and wasting in leg; onset gradual without preceding hemiplegia.* (Under the care of Dr. Ramskill.)—M. P——, æt. 12, was an active and intelligent child until her sixth year, when she became subject to attacks of transient rigidity in the right limbs, at first affecting only the ankle-joint, afterwards the thigh-muscles and knee-joint, and after

some months the right arm also. As the area involved increased, each attack became longer in duration. At first each attack lasted for one or two minutes, but ultimately the rigid state continued for an hour or an hour and a half. A year after the commencement of these attacks she had measles, and after this the spasmodic stiffness of the right side became constant, and her mental state rapidly deteriorated for a few months, then became stationary, and subsequently slightly improved under careful training. The muscular spasm, however, steadily increased. Until the previous spring she had been able to get about a little, she then had a feverish attack and after that became unable to stand.

On two occasions she had been unable to speak for six weeks.

In November, 1870, the following note was made of her condition :—She is able to answer simple questions correctly, though slowly, but the expression of her face is vacant ; when addressed she smiles unmeaningly, and on the slightest incentive laughs and smiles immoderately.

Her features when at rest are equal : the angles of the mouth even. In all emotional movements the right side of the face moves much more than the left, and moves indeed excessively.

The right arm is in a state of continuous slow mobile spasm, variable in degree but never ceasing whilst the child is awake, but ceasing entirely during sleep. The spasm involves the trapezius, latissimus dorsi, infra-spinatus, pectoralis, triceps and biceps, forearm and hand muscles, but not in a uniform manner, varying in different muscles at different times, so that the posture of the limb changes a little. Its usual position is with the wrist flexed, the forearm pronated, the elbow extended and the arm behind the trunk. She is always able to bring the arm to the front of the body, but much more easily at some moments than at others. The head of the humerus seems to be dislocated to the front of the coracoid process, and there to have formed a false joint. Usually the

spasm in the triceps predominates and the arm is extended; but every now and then that in the biceps becomes the greater and the elbow is flexed at a right angle. Occasionally slight jerking spasm can be felt in the rigid muscles.

All movements in the arm cease during sleep and the limb is much less stiff. There is no wasting of the arm : its circumference is the same as that of the left arm. There is no diminution of faradic irritability in the muscles.

In the leg there is no mobile spasm comparable to that in the arm, but considerable permanent fixed rigidity, with wasting of the muscles. The knee is extended; the muscles of the thigh, front and back are moderately wasted, but the calf muscles are greatly wasted and contracted to such a degree of talipes equinus that the normal angle of the ankle is reversed, and the anterior line of the leg and foot forms a convexity. The circumference of the calf is one inch less than the left. Faradic irritability is present in all the muscles of the leg with the exception of the calf muscles, but in all it is considerably less than in the left leg.

There is no impairment of sensation in the right limbs. The left arm and leg are normal.

Little change occurred in her condition while she was under observation. Five years later her father wrote that the movements continued violent, so as to interfere with all use of the limbs. Attempts at movement increased the spasm, and it was increased by movement of the left hand, so that "in eating or working with the left hand she has to put her right hand behind her back and jam it against the chair to keep it still." The contraction in the leg continued, the heel being four or five inches from the ground.

CASE 12.—*Irregular clonic spasm and slighter variable tonic spasm in right arm and leg ; sequel of partial hemiplegia ; paralysis of opposite second and third nerves.*

—H. H—, a girl æt. 12 (admitted into University College Hospital, October 31, 1874, under the care of Dr. Reynolds). Only an imperfect history of her early symptoms could be obtained. It appeared that when three years old an attack of right hemiplegia came on in the night; the arm and leg in the morning being " stiff " and weak, though capable of slight movement. A few weeks afterwards spontaneous movements commenced, similar to those existing on admission. The strabismus was also noticed soon after the onset. For two years she was unable to walk. When four years old the right tendo Achillis was divided with slight improvement in the power of the leg. From time to time the movements varied in intensity, but their character remained unchanged.

State on admission. Mind unaffected; a quick intelligent child. Eyes: right, natural in appearance, pupil medium size, vision $\frac{1}{2}$; left, counts fingers only, pupil large, acts sluggishly to light. No change in field of vision. Divergent strabismus depending on defective motor power of left eye. Movement outwards free, inwards and downwards very slight, upwards less than the right eye. Ophthalmoscopic appearance:—each disc pale, but no decided atrophy; the two are similar. No ptosis. No affection of fifth nerve. Face, lower part slightly weaker on right side.

The head is straight; the sterno-mastoid free from spasm. The right arm is smaller than the other; in length one inch less; in circumference, upper arm and forearm each half-an-inch less than the left. On account of the continual spasm in the arm the hand is commonly held with the left hand. The right shoulder is much higher than the left. If the arm is free to move the position it assumes is with the upper arm straight forwards from the shoulder and almost horizontal, with the elbow flexed so as to bring the hand almost in contact with the face. The wrist is sometimes over-extended, sometimes so much flexed that the metacarpus is bent into arch ; it is commonly pronated; the metacarpo-phalangeal joints are flexed ; the

phalangeal joints also commonly flexed. The limb is, however, rarely still for more than a second, all the muscles are the seat of irregular contractions, which occur suddenly and cease suddenly. The latissimus dorsi, trapezius, infra-spinatus, pectoralis, deltoid, biceps, triceps, flexors and extensors of wrist and fingers, are all involved. Irregular movements result, jerky from the suddenness of the muscular contractions causing them. Sometimes they are so severe as to carry the hand up above the head or strike it against the face. When the sudden spasm completely ceases, the muscles as a rule are not relaxed. Persistent slight tonic spasm remains, keeping the limb in a certain general position. This, however, also, sometimes ceases, and the limb falls to the side with relaxation of the tonic spasm, only very slight clonic movements continuing. After a second or two the severe clonic and tonic spasm recommences; when the limb is thus relaxed not the slightest shortening of any muscle can be detected. An attempt to restrain the clonic spasm increases its intensity. All movement ceases during sleep. There is considerable voluntary power over all the muscles involved. If she tries to execute any movement she cannot at once do it, but after a little time the voluntary action gets the better of the spasm for a few seconds.

The left leg is also smaller than the right in length, 1¼ inch less; in circumference, the thigh ¾ inch, the calf 1 inch less; the foot is ¼ inch shorter than the other.

The common posture is with the hip and thigh a little flexed, but she can extend them at will. Sometimes, and especially when she stands upon it, the limb is still; at other times there are irregular jerky contractions in all the muscles, causing slight movements of increased flexion, and slight movements of the toes.

There seems to be no relation between the movements in the leg and in the arm. There is no spasm of the abdominal muscles. In respiration there is a little less movement in the right side of the chest than in the left.

She was treated with hypodermic injection of morphia

and with the Calabar bean. Slight diminution in the movements resulted.

A year afterwards, she having taken tonics during that time, the movements were decidedly less. There was little spasm in the latissimus dorsi or trapezius, that in the deltoid and infra-spinatus chiefly determined the position of the arm, which was slightly abducted and moved backwards. It still would not rest upon the lap. The elbow was still flexed by the biceps. It was difficult for her to straighten the fingers. Considerable curvature of the spine, convexity to the right, had come on.

Case 13.—*Irregular clonic (choreoid) spasm in left arm after hemiplegia, on movement chiefly.* (Under the care of Dr. Radcliffe, notes taken by Dr. Sturge.)—Mary Anne C—, æt. 52, of good previous health, and without inherited neurotic or other proclivity, was seized with left hemiplegia at fifty years of age, two years before she came under observation (in 1875). The onset was sudden, one evening, without loss of consciousness. The arm became almost powerless, the leg very weak ; she could move it, although she could not stand without assistance. It is uncertain whether the paralysis of the arm was at first complete. She remembers no loss of sensation. At the end of a week she was beginning to move her arm, and one day, in making a violent effort to pick up a small object, the arm suddenly flew up, causing her to throw the object from her with some force. From this moment the arm was the seat of spontaneous irregular movements, and jerked violently up and down, so that she was unable to hold anything. After six weeks the arm was much quieter, the irregular movements occurring only on voluntary motion. The strength of the arm gradually improved. The leg gained power sooner than the arm. At the end of a week she could walk a little, and at the end of six months the leg was fairly well.

On admission it was noted that she was old-looking for

her age. The face, and other parts supplied by cranial nerves, were equal in their movements. The arm was habitually held at the side, and in that position was still, and she could flex and extend the elbow and wrist steadily. If the arm was moved from the side it was the subject of violent irregular movements, choreoid in character, affecting all parts of the arm, the fingers, perhaps, more than the rest. The movement was distinctly increased by attention ; and sometimes, when it was absent, attention would bring it on. She could not hold a fork in the hand. Sensation in the arm was unaffected. The leg was quite steady and fairly strong, and presented no trace of spasm.

A week afterwards the movements were less, so that the grasp of the hand could be tested (right 65, left 40 ko., Charrière's dynamometer). On testing the irritability of the muscles, it was found that in the affected arm there was slight increase in the farado-muscular irritability, but no change to the constant current. She was treated with the constant current to the arm, negative electricity being run off, according to Dr. Radcliffe's method. She left the hospital at the end of three weeks, the arm having become so much steadier that she was able to hold a fork, and do many things with it which she could not do before.

CASE 14.—*Irregular, quick, clonic spasm, and inco-ördination in right arm, after hemiplegia late in life.* (Notes taken chiefly by Dr. Sturge.)—Mary H—, æt. 53. Health good before seizure, except an attack of rheumatic fever ten years previously. No history of syphilis. Two years before she came under observation, on rising one morning, she felt much numbness of the right hand and arm, without at first any weakness. She went down stairs and came up again. On reaching the top she fell to the right, and found she had lost all power in the right arm and leg. She was put to bed, remaining conscious. Articulation was difficult. A few hours afterwards, con-sciousness was suddenly lost, and she remembers nothing for three days. Good articulation had returned in three

days more. There was no aphasia. In three months she was able to move the arm a little, and to stand. Ever since the first powér of moving the arm the irregular movements have been present.

State on admission (in 1874).—There is no mental impairment or affection of speech. The face has a little less power on the right than on the left side. Other cranial nerves are normal. The right arm possesses fair power in all movements. The limb is the seat of continuous, quick, irregular, jerky movements, increased by voluntary motion, and causing jerky incoördination, which makes it almost impossible for her to take hold of any object. The habitual position of the hand is with the wrist slightly flexed, the little finger flexed at the proximal and extended at the distal joints, the others less flexed, and the fore-finger quite straight, extended. All the fingers are separated from one another. The thumb is extended, but adducted, in a line with the forearm. On attempting to close her fist the fourth metacarpo-phalangeal articulation alone is flexed, the others remain more or less extended, but the phalangeal articulations are flexed as in health. The opponens pollicis is almost always strongly contracted, so as to bring the ball of the thumb towards the palm. She cannot coördinate the movements of the fingers so as to hold an object between the finger and thumb, but has to grasp it strongly with her whole hand. The arm is well nourished. Her leg is steady, and she walks with a slight limp, throwing the leg round, although she can raise her toes well when she tries. Sensation to touch on the right side of the face and right limbs is distinctly defective. There is no loss of faradic irritability in the muscles of her limbs. No excessive reflex action in the leg. The heart sounds are normal.

There was little change in her condition during a short time that she remained under observation.

Case 15. — *Extreme incoördination of right arm after hemiplegia ; cicatrix in optic thalamus ; no degeneration in*

cord.—W. J—, a painter, æt. 55, who had never had lead
colic, but was found on subsequent examination to be
suffering from albuminuria, was seized with right hemi-
plegia in May, 1872. He found when painting that the
brush fell out of his hand, which shook a little, and he had
a feeling of tingling in his hand and arm. He tried to
go on working, but the arm gradually became weaker,
and the leg also became weak, so that in about half an
hour he had to leave off work. He was assisted home,
walking a short distance. On the way he spoke indis-
tinctly, "mumbling." He had to be carried indoors, and
states that immediately afterwards his head became turned
to the right, he became unable to speak, and lost
consciousness. He remembered nothing more till the
next day; then he could move the arm and leg a little,
but could not hold objects; he could swallow, but spoke
with some difficulty, not, however, misusing words.
Power soon improved, and in a week he was able to walk
a little. As the arm regained power its movements
became unsteady.

He was first seen three months after the onset of the
hemiplegia. There was then no noticeable paralysis of
the face or tongue. The leg had recovered to a large
extent; he could walk several miles, and it was
quite steady. The arm possessed considerable power
although it was weaker than the leg. At rest the hand
was perfectly steady, but on voluntary movement, violent
jerking spasm occurred, producing a wild incoördination,
intermediate in character between that of chorea and of
cerebro-spinal sclerosis. The ataxy was so extreme that
if he attempted suddenly to raise an object from the table
the hand would fly up over his head.

For a time this ataxy remained stationary, but after a
year, while taking first arsenic, and subsequently bromide
of potassium and quinine, the hand became gradually
much steadier. He was seen occasionally, and three years
after the onset it was noted that the incoördination had
to a large extent disappeared: he could lift a glass of

water three parts full without spilling it, but not one quite full. He could even write a little, grasping a pencil very tightly and forming the letters rapidly, but he complained that while doing so he could feel the arm "jumping." Power was nearly as good as the left; (dynamometer, left 20 ko., right 18 ko.). He had some difficulty in relaxing his grasp.

Unfortunately no note was made as to the sensibility of the limbs, but it was noted that he had no defect in his field of vision.

The patient died shortly afterwards from his renal disease. A partial post-mortem examination was made, under circumstances of difficulty, three days after death. A careful examination showed but one lesion in the brain, and that was situated in the left optic thalamus. There was a transverse depression on the upper surface of the thalamus which passed from the inner side just in front of the posterior tubercle (pulvinar) outwards and a little forwards to the outer part of the thalamus opposite its middle. The depression was somewhat irregular, $\frac{1}{8}$th inch wide, and $\frac{1}{12}$th deep. Beneath it there was found, on section, a puckered cicatrix, which passed through the thalamus from one side to the other. It consisted of a hard reddish-white centre, the cross section of which was about the size of a mustard seed, from which lines of fibrous tissue passed. It was separated from the bottom of the depression above it by some healthy tissue. This cicatrix was lower at the outer part, where at its termination it lay immediately above but did not involve the layer of white fibres from the crus (of the corona radiata). On subsequent microscopic examination it was composed of fine nucleated fibrous tissue, mingled with masses of granular pigment, and was separated from the adjacent nerve tissue by a zone of extremely dilated vessels.

No lesion existed in the cerebellum. The crus, medulla and cord were all free from any descending degeneration. There was a good deal of connective tissue in the cord, and perhaps rather more in the right anterior column than

in the left, but the lateral column and the posterior column were both free from relative excess. The nerve cells in the anterior horn were distinct, numerous, and apparently healthy.

CASE 16.—*Simple ataxy of right arm after hemiplegia.*—G. B—, plumber, æt. 55, had never suffered from lead colic, but had had one attack of gout. His urine contained a large quantity of albumen, granular casts, and had a low specific gravity. He was attacked with hemiplegia a year and a half before he came under observation (in November, 1872). For some years he had been troubled with headache, and during the year before the attack he had suffered about once or twice a month from attacks of " numbness " in the right finger and thumb, each attack lasting ten or fifteen minutes. The onset of the hemiplegia was in the night. He remembers nothing after going to bed, but was heard, about 4 A.M., to fall out of bed, and was found unconscious on the floor and paralysed on the right side. Next day he was partially conscious. The sphincters at first were inefficient. There was some thickness of speech and difficulty of swallowing. He frequently called objects by wrong names, asking for " scissors " instead of a " plate." In a fortnight some power returned to his leg and in a month he could walk. In two months from the onset his arm began to improve. As soon as he began to move the arm he observed the same unsteadiness which existed when seen a year and a half later.

Slight weakness of the arm then continued; the grasp of the left hand was 40 kilogrammes, that of the right 13 only. The arm could be moved freely in all directions; there was no permanent rigidity or spontaneous spasm, but great inco-ordination of voluntary movement. On trying to execute any act with it, it swayed about, the fingers sprawling, and it was almost impossible for him to pick up with them any small object. With his eyes shut the disorder of movement was still greater; he could just

manage to touch his nose with his forefinger when his eyes were open, but when his eyes were shut he could not do so, and the hand went wandering about on one side or the other. There was no difference in the electric irritability in the two arms.

His leg possessed much more power than his arm, and he could walk several miles but with a limp. There was no irregularity of movement with the eyes open or shut, but it was noticed that the toes were raised rather high. There was slight paralysis of the right side of the face (zygomatic muscles) and he could close the right eye alone, but not the left one. Before the attack he could close either eye, separately. The tongue was protruded straight.

In the whole right side tactile sensibility was distinctly defective, but sensibility to pain was normal.

His sight was dim, in consequence of retinal hæmorrhage secondary to his renal mischief.

This condition of the right limbs continued as long as he remained under observation. Some months after he was first seen, suddenly "numbness," defective sensibility to touch, appeared in the left arm, which cleared, except in the thumb and forefinger, where it persisted as complete anæsthesia over the last two phalanges of the thumb (palmar aspect) and adjacent side of the forefinger, the sensibility on the ulnar side of the forefinger being normal. Sensibility to pain persisted. At the same time there was some loss of tactile sensibility in the lips for half an inch around the mouth; taste was little impaired, but the sense of smell became almost lost and a curious condition of spasm appeared in the right platysma myoides. These symptoms persisted until his death, a few weeks later, which unfortunately occurred away from the hospital and a post-mortem examination could not be obtained.

CASE 17.—*Intermitting clonic spasm of left hand-muscles, on movement only ; the sequel of hemiplegia, probably due to disease of pons Varolii.*—Edward P——, an artist, æt. 55,

showing much senile degeneration, but having no heart disease, was seized with left hemiplegia in July, 1873. For two years before, he had been liable to attacks of giddiness. One morning, when eating his breakfast, he found that his jaw had closed firmly, and he had bitten his tongue. He tried to force his finger into his mouth but could not. After a time this spasm became slighter, but it subsequently returned, and recurred several times in the course of the day. Next morning he felt well on rising, but had a difficulty in getting down stairs, and almost immediately was seized with "trembling" and his face became "drawn." A doctor who was sent for said that the left side of the face was paralysed. In the evening it was found that the right arm and leg were weak. There were no absolute paralysis. Recovery of power was gradual. Articulation was indistinct for about seven weeks and he used words wrongly, asking, for instance, for a brush when he wanted canvas.

Two years after the onset of the hemiplegia the following note was made :—The face moves distinctly more on the right side than the left. There is no affection of any other cranial nerve. The masseters contract equally, the tongue is protruded straight. He can move the right arm readily, but it is still the weaker (right 15 ko., left 30 ko.). Apart from voluntary movement the arm is quiet and has no rigidity. Grasp not very steady : he cannot bring the first finger up to the others without assistance. On holding the hand out, slight irregular contractions can be felt in the muscles of the arm, and the fingers are the seat of rapid alternating movements of extension and flexion, abduction and adduction, all chiefly at the meta-carpo-phalangeal joints, and apparently due, in part, to intermitting spasm in the interossei. The little finger moves more than the rest, the first finger least. The movement reminds one of the "tambour beating" of paralysis agitans, but the movements are more extensive in range and less regular, alternating in intensity in different fingers. The thumb is flexed and extended

somewhat irregularly. There is a little motion of the wrist-joint, flexion and extension, abduction and adduction. There is very little incoördination of voluntary movement; he can button or unbutton his clothes easily. On rest the hand is at once still.

There is no tremor of the foot even on exertion.

CASE 18.—*Continuous regular clonic spasm in fingers of left hand (due to interossei), after hemiplegia; similar more widely-spread spasm on movement.*—James J. P—, æt. about 50, was suddenly seized with right sided hemiopia, which came on with pain in the head and transient loss of consciousness. Two months afterwards an attack of imperfect left hemiplegia occurred. It is doubtful whether consciousness was lost at the onset but there was considerable mental excitement. Three weeks afterwards, when he was kindly sent to me by Dr. Mitchell Bruce, the hemiopia continued, the face and tongue were free from paralysis. The left arm was weak and its movements were slightly unsteady. There was no marked incoördination, but slight clonic spasm occurred as he raised it to his head. The leg was also weak but steady. He had loud mitral and aortic regurgitant murmurs.

Two months afterwards he was seen, through the courtesy of Dr. Ligertwood, in the Royal Hospital, Chelsea. The hemiplegic weakness was about the same. The slight clonic spasm, on raising the arm, continued. In addition there were certain movements when the hand was at rest.

The third and fourth fingers were slightly flexed at the metacarpo-phalangeal joints and extended at the others and underwent constant movements of abduction and adduction, each movement occurring once or twice a second. The movements were evidently due to the action of the interossei. The second finger was scarcely moved. On any change of position of the hand, or on an effort to restrain the movement, it ceased for a moment and then recommenced. The thumb and forefinger, when not moved voluntarily, were perfectly still : but if moved, as in

an attempt to pick up an object, the voluntary movement was modified by clonic spasm precisely similar to that which was continuous in the other two fingers,—the thumb and forefinger being alternately approximated and separated. The leg at rest and movement was free from spasm, except when the toes were resting on the ground, when there was a little jerky tremor in the limb such as occurs in paraplegia, and has been associated by Charcot with the secondary degeneration in the cord. The hemiopia continued unchanged.

He died about twelve months later. No post-mortem examination was obtained.

DESCRIPTION OF PLATE XII.

Figs. 1 and 2.—Hand in Case 1 of slow movements like "athetosis," following slight hemiplegia. The hand could only be kept still enough to be photographed in these two postures, but was in constant slow movement in intermediate positions.

Fig. 3.—Foot in same case, showing its inversion and the spasmodic contraction of the short extensor of toes.

Fig. 4.—Hand in Case 4. Mobile and tonic spasm after hemiplegia. Showing position determined by contraction and spasm in interossei.

Figs. 5 and 6.—Positions assumed on two successive attempts to extend fingers (same case).

Fig. 7 (Case 3).—Fixed and mobile spasm in arm after hemiplegia in early life. Hypertrophy of muscles of shortened limb. Over-action of muscles of face on same side.

Figs. 8 and 9 (same case).—Changes of position (involuntary) in hand. In fig. 8 the thumb is held apart to show the over-extension of the middle phalanges of the fingers. The posture shown in fig. 9 is that assumed by the fingers as soon as the thumb was released.

Fig. 10 (Case 2).—Irregular position and involuntary movement of fingers, due chiefly to interossei.

Plate XII

Fig 1

Fig 2

Fig 3

Fig 4

Fig 5

Fig 6

Fig 7

Fig 8

Fig 9

Fig 10
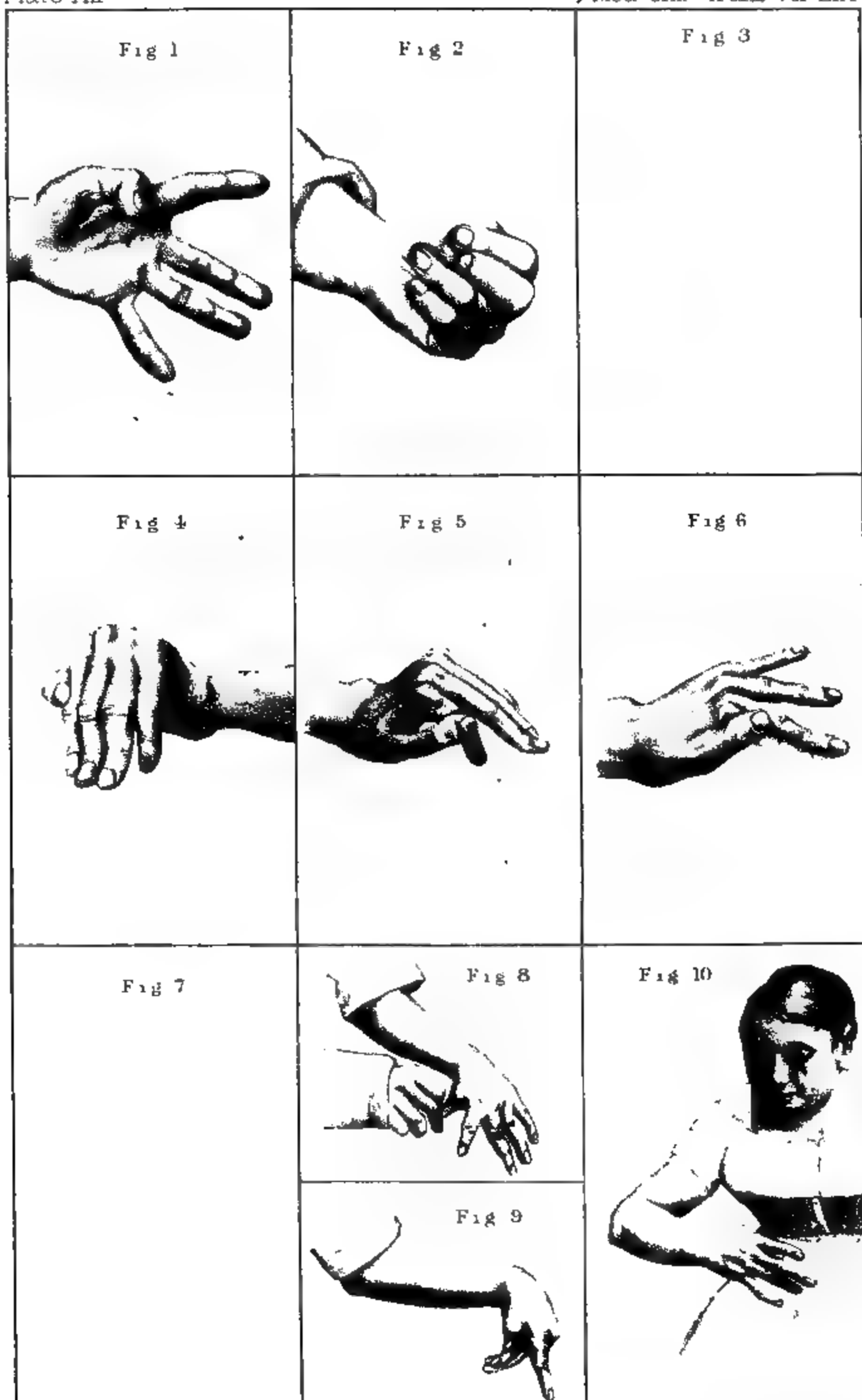
ON THE

NATURE AND MODE OF ORIGIN

OF THE

LEAD LINE IN THE GUMS.

BY

C. HILTON FAGGE, M.D., F.R.C.P.,

ASSISTANT PHYSICIAN TO, AND LECTURER ON PATHOLOGY AT,
GUY'S HOSPITAL,

MORE than thirty-four years ago, Dr. Henry Burton, in a communication to the Royal Medical and Chirurgical Society, pointed out that in persons under the influence of lead a peculiar blue line could be discovered on the borders of the gums. Mr. Tomes subsequently gave a tolerably complete explanation of its production. But I am not aware that the microscopical appearance of the gingival structures when affected by the metal has hitherto been demonstrated. And having recently had opportunities in two cases of investigating after death the nature of the change, I think it may be interesting to the Society to have my observations brought under their notice, particularly as I believe that they afford grounds for defining, more accurately than has hitherto been possible, the conditions under which the lead-line is developed.

Another circumstance which makes me desirous of drawing the attention of the profession to the subject is the fact that some of the most recent writers on medicine give descriptions of the affection of the gums caused by lead which seem to me inaccurate and misleading. Thus an eminent physician, lately deceased, spoke in his lectures (published in the 'Lancet' in 1872) of " a bluish-purple line next the teeth, and encroaching on them ;" and Dr. Aitken in his well-known text book says that the metal produces ulceration of the gums and alveolar processes, accompanied by a peculiar blue line . . . along the free margin of the gums. And further on, he remarks that " Many pathologists are inclined to believe that the line is owing to the presence of lead in some peculiar state of combination, as with some of the constituents of the tartar of the teeth." Dr. Burton, however, described the line as on the gums themselves ; and he expressly stated that "there was no invariable tumefaction, softening, or tenderness about them," while in another place he seems to have implied that when the gums were ulcerated, tumid, or detached from the teeth by incrustations, such conditions were due to neglect, rather than to the metal.

One character of the lead-line, which I have never seen mentioned in print, was some years ago pointed out to me by Sir W. Gull ; namely, that it is not continuous, but consists of a series of dots arranged side by side at pretty uniform distances. He used to say that the dots corresponded with vascular points which exist in the normal mucous membrane ; and any one who will look at the gums of a few healthy individuals will find that they often present a row of reddish spots, which are precisely similar in position and in size.

The method which I have adopted in examining the lead-line after death has been to slice off a thin layer of the tissue of the gum at the margin next the teeth, and place it beneath the microscope, using a rather low objective. The appearances which are then seen are

represented in a drawing which accompanies the present paper, and which Mr. H. Clarke has kindly made for me. The discoloration is not uniform, but is distributed in the form of rounded loops, some of which look like minute papillary processes of the deeper layer of the mucous membrane, while others seem to be portions of blood-vessels. Under a higher power the pigmentation is seen to be caused by the presence of granules, which may be scattered at a little distance from one another, or collected into dense masses. I have satisfied myself that these are sometimes situated in the interior of the smallest blood-vessels, and sometimes outside them in the tissue immediately adjacent to their walls.

When the lead-line is well marked it may regularly surround the bases of all the teeth. But it is perhaps more commonly imperfect in its development, and then there may be merely two or three dots here and there; generally, these are to be seen in the little processes of gum which project between the teeth, rather than in the notches which correspond with their centres. On the other hand it sometimes happens that the discoloration is present in what may be termed an exaggerated degree. The margins of the gums may then be uniformly blackened, no separate dots being discoverable; and the staining may extend over the mucous membrane covering the whole of the alveolar process, and even show itself upon that which lines the lips and the interior of the cheeks. In the summer of 1875 a patient was in Guy's Hospital under my care, in whom the inner surface of the lower lip presented a black zone of considerable depth. Some observers have thought that the teeth themselves may become blackened from the same cause; and Mr. Tomes speaks of the tartar as being similarly discoloured, especially where it is in contact with the gum. But I hardly think that this can be the case; and I should be disposed to regard any such appearances as accidental, since they are by no means uncommon in persons who cannot be supposed to be under the influence of the metal.

However this may be, it is clear that the so-called blue line is itself really *in* the gum and not *on* its surface. And the blue colour seems to me only apparent. When examined with a lens during life, the dots which make up the line are seen to be black. My friend, Dr. F. Taylor, has suggested to me that the reason why as a whole it looks blue is that much of the pigment is seen through a comparatively thick layer of translucent tissue. There are many other examples of black pigments which seem to be blue when embedded in or beneath similar fibrous textures.

Mr. Tomes some years ago suggested that the colouring material in the blue line on the gums was probably a sulphuret of lead, and he pointed out that the tartar about the teeth is so porous as "readily to admit into its substance fluids charged with animal matter, which may there be decomposed and furnish sulphuretted hydrogen." · We are now able, I think, to form a still more accurate conception of the mode of production of the line than seems to have suggested itself to Mr. Tomes. We see that the sulphuretted hydrogen must diffuse itself into the gingival tissues, and combine with the lead as it is actually circulating in the blood which passes through the vessels of the gum, or as it is contained in the plasma which oozes out of the vessels for the nourishment of the epithelial and other structures. If the lead were previously in combination with the elements of the connective tissue of the gum, the line would not to the naked eye appear dotted; nor would it under the microscope present the loops and serpentine coils shown in the drawing.[1]

It appears to me probable that the sulphuretted hydrogen is often derived directly from portions of meat or of other animal food, which may have become wedged in the angles between the teeth, and undergo decomposition there. For, as I have already remarked, when the line is

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[1] My observations are, of course, incompatible with the view advanced by Naunyn in von Ziemssen's 'Handbuch' (vol. xv, p. 263), that the lead probably enters the oral cavity from without in the form of particles of the metal. (Oct., 1876.)

imperfect the dots which represent its rudimentary condition are often limited to the processes of gum which project upwards into those angles.

Mr. Tomes offered, as an alternative, the suggestion that the sulphocyanogen of the saliva might possibly furnish the sulphur for the production of the line. But I think that this is negatived by the limitation of the line to the margin of the gum close to the teeth, and by the fact (which Mr. Tomes pointed out), that wherever there is a gap in the row of teeth the line is wanting. The influence of the saliva would be more equally distributed over all parts of the gums.

It appears, therefore, that the lead line may be regarded as a *precipitate* of the metal from the blood, or even, in some sense, as an *excretion*. This view affords an explanation of several points in regard to the line which have hitherto appeared of doubtful interpretation. One is the fact, noticed by Mr. Tomes, that it is sometimes seen in the gums of persons who are not known to have been exposed to the influence of the poison, and who have had none of its symptoms. Mr. Tomes himself suspected that other metals, besides lead, might sometimes give rise to a similar discoloration.[1] The late Dr. Brinton considered that in some patients who had taken bismuth he had seen an affection of this kind. But it appears evident from his brief description that he was referring to the reddish-purple border which is commonly present when the gums are in an unhealthy state, and which is altogether different, being simply due to vascular injection. If any other metal were really capable of producing a line resembling the lead line, I think that the fact must before this have become fully recognised. It seems to me probable that whenever the appearance in question is observed, even though the person may present no other indication of having been affected by the poison, it is really caused by lead, but that this metal has in such cases been taken into the body in quantities too small to give rise to symptoms.

[1] 'Brinton on Ulcer of the Stomach,' 1857, p. 118.

The black line being a precipitate of sulphide of lead
from the circulating fluid, one can see how it might be
produced in course of time by the action of sulphuretted
hydrogen on the blood constantly flowing through the
gingival vessels, although the amount of the metal present
in the body at any one moment might be almost infini-
tesimally minute, and even although the total amount pre-
sent, during all the time in which the line was in process
of formation, might be quite inconsiderable.

It is, indeed, a difficulty which stands in the way of the
acceptance of this hypothesis, that the lead line in the gums
seems not to be permanent, at least in many instances.
Dr. Burton mentions that in a few of his cases it had actu-
ally disappeared before the patients left the hospital. Pro-
bably it may pass off, if the absorption of the metal ceases,
even though the internal structures may still retain the
deposits of lead, which are known to occur in those who have
been exposed to its influence. I do not know in what
other way one can explain the fact that the administration
of the iodide of potassium to a person who has been
poisoned by lead, but who is placed under conditions
which no longer involve its entrance into the body, is
sometimes followed by the development of a line on the
gums which had previously been wanting. I have observed
this on three occasions at least, and Dr. Frank Smith, of
Sheffield, has independently noticed it. It is strictly
parallel to the well-known circumstance that when iodide
of potassium is given to a patient affected with chronic
plumbism, but withdrawn from the direct influence of the
poison, the urine begins to contain lead, although none
could before be discovered in it. The only difficulty is to
understand how the lead line came to be absent until the
iodide began to be administered. But this difficulty is
removed by the supposition that it really had existed at
one time, but had disappeared during the interval that had
elapsed since the metal ceased to be absorbed.

Another conclusion, which is deducible from the view
suggested by Mr. Tomes with regard to the line in the

gums, is that in a person who keeps his teeth clean it may be wanting even though he may be really suffering from lead-poisoning. I have long made it a rule to attach no importance to the absence of the line in individuals with very clean teeth and gums; but I cannot cite any case in which my caution was justified by the result. I have, however, more than once told a patient to leave off cleaning his teeth when he was taking iodide of potassium, in order that the conditions might be favorable for the development of the line.

It ought also to follow from Mr. Tomes' theory that the sulphide of lead should be deposited in all other parts of the body which are exposed to the influence of sulphuretted hydrogen. This gas is often present in the intestine, the mucous surface of which ought accordingly to be blackened, at least as much as the gums. I am not aware that this has hitherto been noticed; but it is possible that the frequency with which the intestinal lining membrane is found discoloured under other circumstances may have prevented observers from attaching any importance to such an appearance, even if it should have been present. In November, 1875, I made an autopsy in the case of a man, æt. 43, a painter, who had had colic fifteen years back, and who, while in the hospital, had complained of pain about the splenic flexure of the colon. I found in this part of the bowel, as well as in the transverse colon, some scattered cicatrices, with puckering of the mucous membrane. They were slate-coloured, but I did not at the time notice that their appearance differed from that of the scars which are found after ulceration from dysentery or other causes.

DESCRIPTION OF PLATE XIII.

LEAD LINE IN THE GUMS.

Thin slice of gum affected with the lead line. Magnified 200 diameters.

(From drawing by Mr. H. Clarke.)

Plate XIII

ON INGRAVESCENT APOPLEXY.

A CONTRIBUTION TO THE LOCALISATION OF CEREBRAL LESIONS.

BY

WILLIAM H. BROADBENT, M.D.,

PHYSICIAN TO, AND JOINT LECTURER ON MEDICINE AT ST. MARY'S HOSPITAL;
PHYSICIAN TO THE LONDON FEVER HOSPITAL.

SIR THOMAS WATSON following Abercrombie and corroborating by his own observation the distinctions laid down by Abercrombie describes three forms of apoplectic attack.

" In the first form of the attack, the patient falls down suddenly, deprived of sense and motion, and lies like a person in a deep sleep; his face generally flushed, his breathing stertorous, his pulse full and not frequent, sometimes below the natural standard. In some of these convulsions occur; in others rigidity and contraction of the muscles of the limbs, sometimes on one side only.

" Now, respecting persons seen in this condition, the immediate prognosis is uncertain. Some die in a short time and much blood is found extravasated within the cranium. Some die after a rather longer interval, and

then we often find serous effusion only. And in some
that die early, no effusion either of blood or of serum can
be detected. Some recover altogether without any ill
effect of the attack remaining. Others recover from the
coma, but are left paralytic of one side, and with some
imperfection of speech, or of one or more of the senses.
And this paralysis and imperfection may disappear in a
few days, or gradually depart, or remain for life.

In the second form of attack, the coma is not the
earliest symptom. The disease generally begins with
sudden and sharp pain in the head. The patient becomes
pale, faint, and sick, and usually vomits, and sometimes,
but not always, falls down in a state of syncope, with a
bloodless and cold skin, and a feeble pulse. This also is
occasionally accompanied by some degree of convulsion.
Sometimes he does not fall down, the sudden attack of
pain being accompanied by slight and transient confusion.
In either case he commonly recovers in a short time from
these symptoms, and is quite sensible and able to walk ;
but the headache does not leave him. After a certain
interval, which may vary from a few minutes to several
hours—and Dr. Abercrombie records cases in which it
was even much longer—the patient becomes heavy, for-
getful, incoherent, and sinks into coma, from which he
never rises again. In some instances, paralysis of one
side occurs ; but perhaps more often, there is no palsy
observed.

The disease when it comes on in this way, is much
more uniform, and of much worse omen, than when it
commences after the former fashion. Upon inspection,
says Dr. Abercrombie, we find none of those varieties and
ambiguities, which occur in the apoplectic cases, the coma
being present from the first ; and the second class which
we are now considering, he calls cases not primarily
apoplectic.

The third form of attack described by Dr. Abercrombie
can scarcely be said to be an apoplectic attack at all ;
indeed he himself includes this form in the class of

paralytic cases. It is characterised by sudden loss of power on one side of the body, and frequently by loss or impairment of speech, without loss of consciousness."

To the second form of attack the name Ingravescent Apoplexy has been given. This I now proceed to con-consider. Its features are the absence of loss of consciousness at the outset, the gradual accession of symptoms, the almost invariably fatal result, and the large amount of blood found to be extravasated. I ought to add that vomiting invariably occurs at an early period of the case. My observations show, that in these cases not only is the hæmorrhage large, but that its situation is within certain limits constant, and I believe that an anatomical explanation can be given of all the phenomena characteristic of the attack.

The seat of the extravasation is on the outer side of the extra-ventricular corpus striatum (Linsenkern, lenticular nucleus) between this ganglion and the external capsule, a plane of fibres which separates it from the convolutions of the island of Reil, and it has appeared to me that the large amount of hæmorrhage is explained first by the size of the vessel or vessels ruptured, and secondly by the very slight resistance to extravasation. Also that the absence of loss of consciousness in the early stage of the attack is explained by the fact, that until the quantity of blood poured out is considerable there is no extensive rupture of fibres or ploughing up of grey matter, and probably no compression of brain substance.

A hemisphere in which hæmorrhage has occurred in the situation and to the amount here in question will present the usual external appearances suggestive of internal pressure, flattening of the convolutions, exclusion of blood from the minute vessels on the ridges, and obliteration of the sulci. If the examination is made simply by the usual sections many interesting points will escape observation. The fissures of Sylvius should be freely opened out, when less moisture will be found in that of the side on which the lesion has occurred than in its fellow, and the pia mater

will contain less blood; more remarkable, however, will be
the condition of the convolutions of the insula, which will
be flattened out and obliterated, so that the island of Reil
has the appearance of a smooth oval elevation. Usually
an incision into it at once exposes the clot. Occasionally,
indeed, the thin layer of nervous matter over the effused
blood is torn in the examination, especially when the clot
extends under the anterior perforated space. I have
never seen hæmorrhage into the fissure in these cases,
though I have known the blood enter and fill the ven-
tricles, burst through the floor of the third ventricle into
the interpeduncular space and travel beneath the arach-
noid to the Sylvian fissures, finding its way far into the
one on the opposite side from the original lesion, but being
excluded by pressure from the one near which it was
originally effused.

After exploration of the Sylvian fissures the examina-
tion may be continued by the usual sections from above
downwards. The blood may have penetrated the ven-
tricle or it may have ploughed up the hemisphere on the
outer side of the ventricular cavity. Occasionally in the
latter case it even reaches the surface of the brain; in
the only instance in which I have seen this, the blood had
split up the ascending gyri on each side of the sulcus of
Rolando (it never, so far as I know, escapes into the bottom
of a sulcus). In either case, whether the clot is intra or
extra ventricular, unless special care be taken it will pro-
bably be concluded that the corpus striatum is destroyed,
or at least torn by the blood, whereas it will usually be
found entire and intact, or only penetrated by a process
of the clot, but completely dissected off from the
hemisphere proper and displaced inwards. A detail of
some little interest may here be added, namely, that when
the blood has entered the lateral ventricle, while all the
ventricles may be filled and the cornua distended by clot
and fluid, the descending cornu on the side of the lesion
will be all but empty, the blood being excluded from it by
antecedent pressure on it by the earlier extravasation.

It will now be necessary to refer to the anatomy of the part of the brain in which the lesion occurs, and to describe in some detail the relation of parts, the course of fibres, and the distribution of blood-vessels. For this purpose drawings of sections of the brain are shown, which I venture to interpret from my investigations on the course of the fibres of the hemispheres.

The internal capsule, as it has been absurdly called, is constituted by the ascending and diverging fibres which enter the cerebrum below as the crus between the extra-ventricular corpus striatum and thalamus, and emerge above from the region of the central ganglia to enter the hemisphere proper as the corona radiata. If any fibres of the crus pass uninterruptedly to the hemisphere it is in this tract. As the crus passes into the interval between the ganglia its two divisions, presumably motor and sensory, the crusta and tegumentum, are distinct one from the other, but this separation is soon lost and in the corona radiata there is a complete intermingling of fibres, so that no distinction can be made between those which in origin and function are different. We are not here concerned with the rearrangement of fibres which takes place as the crus becomes corona radiata, but the increase in number is obviously very great. To this increase the thalamus contributes conspicuously, the corpus striatum less conspicuously, though probably more abundantly. It is not easy to trace fibres arising in the intra-ventricular corpus striatum and proceeding onwards to the hemispheres, but the radiating fibres in passing between the head of this ganglion and the extra-ventricular division of the corpus striatum separate into bundles between which the soft grey matter of the two portions dips in and comes into communication, probably giving off fibres. Between these bundles also M. Duret has followed to the intra-ventricular corpus striatum arteries which enter at the anterior perforated space, and far more numerous and larger veins which pass from the extra-ventricular corpus striatum and the hemisphere towards the ventricle, to appear there as the venæ Galeni.

The external capsule will also require a detailed description. It is seen in the various sections as a thin layer of white fibres separating the grey substance of the corpus striatum externum from the cortex of the insula, and it includes a plane of grey matter, the *claustrum*, which appears in the drawings as a thin streak. The convolutions of the island of Reil lying upon the outer wall of the corpus striatum have absolutely no connection with this ganglion, and the claustrum and all outside of it can be removed without disturbing a cell of the ganglion or a fibre arising in it. The corpus striatum then has only a very thin lamina of fibres of its own, forming its proper capsule externally; an equally thin lamina of fibres arising in its cells encases it on its inferior aspect. About these further particulars must be given.

When by careful dissection the proper wall or capsule of the extra-ventricular corpus striatum has been exposed, the temporo-sphenoidal lobe will almost have disappeared and the fissure of Sylvius will be converted into a wide hollow or groove curving round the ganglion which, as the brain lies base upwards, has the appearance of a smooth elevation embracing the crus. The fibres of distribution forming this capsule radiate forwards, backwards, and outwards in all directions from two patches of the grey substance of the ganglion which have become exposed at the summit, one beneath the uncinate lobule, the other just at the outer side of this point. Those passing outwards, forming the proper " external capsule," instead of crossing the Sylvian hollow, as would be expected from the apparent continuity of fibres across it, to the remains of the marginal convolutions of the fissure, dip in between other larger masses of fibres which are those of the corona radiata emerging from the " internal capsule," so that a crossing or intersection here takes place almost at right angles.

A more important anatomical fact relating to this plane of fibres forming the external capsule is that, as Duret has shown, the large arteries which enter the hemisphere at the anterior perforated space for the nutrition of the corpora

striata pass upwards between the capsule and grey matter. To this further reference will be made in the concluding remarks.

CASE 1. *Hemiplegia of left side. Lateral deviation of head and eyes. No loss of consciousness. Sensation greatly impaired. Vomiting. Sleep or stupor. Seven or eight hours after the attack, sudden access of stertor and profound coma. Death in twenty minutes.—Hæmorrhage between the grey substance of the corpus striatum externum and the plane of fibres separating it from convolutions of insula cutting off hemisphere from central ganglia, and ultimately bursting into lateral ventricle. Pulmonary, splenic, and hepatic hæmorrhages.*

This case, though not the first of the kind observed, is placed first because I was able to watch it from an early period of the attack, and to recognise the seat of the lesion.

Wm. S—, æt. 50, foreman of some lime-works in the immediate neighbourhood of St. Mary's Hospital, was admitted into the hospital about 2.15 p.m. on Feb. 9th, 1871, and at once seen by me.

He was an intelligent, steady, sober man, had never had any serious illness, but had lately suffered occasionally from headache, and his appetite had been variable.

On the above-named day he had just gone to work after dinner, when he felt giddy and sat down. He appeared to be confused, but never lost consciousness. Within a few minutes he was seen by a friend to whom he said he felt giddy, but should soon be better; his friend, however, noticed that his head seemed to be turned to the right, that the left arm hung powerless, and that he did not appear to know he had a left arm.

He was at once brought to the hospital, where he arrived about 2.15 p.m. and, being at hand, I was called to him.

He answered questions, gave an accurate account of the

attack, and expressed anxiety about some money which
was in his purse, but his manner was excited and he
called his friend by name and gave him directions, though
he was no longer present. He tried also to take hold of
the paper on which notes were being made, and resisted
when his hand was restrained.

The face was not perceptibly distorted, scarcely so even
when he spoke, and he could whistle though not so well as
usual. The head was turned so that the face looked over
the right shoulder, and the eyes were carried to the
extreme right of the palpebral fissures, the deviation of
head and eyes being most marked. He was unable to look
straight before him, still less to the left.

Left arm and leg quite powerless and relaxed; there
was slight reflex action when the sole was tickled.

Sensation was greatly diminished in face, body, arm,
and leg of this side.

Face slightly flushed; arcus senilis present; pupils
natural.

Pulse 108, rather long and firm, but not strong. Heart-
sounds normal; no reduplication; no evidence of hyper-
trophy. Respiration natural.

A tendency to sleep gradually manifested itself, and
having by careful observation come to the conclusion that
the case was one of those in which a fatal termination is
almost certain and in which a large clot is invariably found,
I determined to bleed. My resident medical officer, Mr.
Boone (now of St. Kitts), opened in succession three veins,
two in one arm and one in the other, but was unable to
obtain more than a very trifling flow of blood. That the
veins were really opened was shown by passing a probe
along them upwards and downwards. I then proposed to
bleed from the jugular, but it was impossible to bring about
the necessary fulness of the vein. While considering the
desirability of dividing the temporal artery the condition
of the patient underwent a change, the face became
bedewed with perspiration, the pulse was softer, and very
shortly he vomited violently, bringing up large quantities

of undigested food, and during the effort urine and fæces were passed involuntarily. He repeatedly provoked fresh acts of vomiting by putting his fingers into the throat in spite of remonstrances. This was about 2.45 p.m.

I did not think it desirable to persist in the attempt to bleed, and a dose of calomel and castor oil was ordered, cold being applied to the head.

The patient continued to sleep during the afternoon, but could be roused, and when awake was quite sensible; he took a little milk and vomited occasionally.

At 10 p.m. stertor suddenly set in, and he was found to be quite unconscious. The stertor continued though he was turned on his side as recommended by Dr. Bowles, of Folkestone (a proceeding which is usually perfectly successful in eliminating the element of danger which stertor adds in these cases), and the respiration was catching. The heart was acting violently, at the rate of 150 beats per minute. During this final attack the lateral deviation of the head and eyes ceased. This was observed for fifteen minutes. The right pupil was large, the left small.

At length, twenty minutes after the stertor set in, the breathing suddenly stopped, but the heart continued to beat and the pulse could be felt for seven and a half minutes afterwards, becoming gradually more feeble. At the moment of death the left pupil dilated.

Post-mortem examination.—Brain,—a little flattening of convolutions of right hemisphere; a superficial, slight, meningeal hæmorrhage of the anterior extremity of frontal lobe on this side. Blood seen to have issued from fourth ventricle, between medulla and cerebellum.

Atheromatous patches in arteries, generally with dilatations.

When fissures of Sylvius opened out, convolutions of island of Reil on right side seen to be flattened out; the entire insula being large and soft.

When hemispheres sliced nothing abnormal met with above level of corpus callosum. When lateral ventricles opened, blood-stained fluid and a small clot found in left,

a rupture in septum lucidum; blood in third ventricle, iter, and fourth ventricle, and in all the cornua of both lateral ventricles. In right lateral ventricle a large black clot, and this being removed a fissure seen (to the outer side of the corpus striatum and thalamus) extending along the entire length of the ventricle, crossed by venæ Galeni and processes of the ependyma left untorn. From this fissure, which was wide and gaping, black clot protruded, on removing which a cavity was found, two and a half or three inches long, which extended along the entire length of the two central ganglia on their outer side. The outer wall of this cavity was for the most part formed by the plane of white fibres which, arising from the grey matter of the corpus striatum externum, separates this grey matter from the convolutions of the insula (external capsule). The inner wall was formed mainly by the grey substance of the extra-ventricular corpus striatum, which was not ploughed up or penetrated by the blood or softened, but was left entire, and was simply dissected off from the fibres. The lower edge ran parallel to the descending cornu of the ventricle along its whole length, and was close to it; superiorly the rupture into the ventricle had taken place by tearing of the fibres of the corona radiata issuing from the central ganglia (internal capsule).

The hæmorrhage had taken place from vessels running from the artery in the Sylvian fissure to the ganglionic substance of the corpus striatum, between the grey matter and the plane of fibres arising from it to pass to the hemisphere. The blood had followed this line of cleavage, and after separating the fibrous capsule from the grey substance had torn through the corona radiata and burst into the ventricle.

Heart not large; valves fairly healthy.

Lungs.—Extensive hæmorrhage into both, the blood black and coagulated; the extravasations had neither the appearance nor distribution of ordinary pulmonary apoplexy.

Considerable hæmorrhage had taken place into substance of spleen, and small scattered hæmorrhages were found in the liver.

Kidneys healthy.

I am of opinion that the pulmonary, splenic, and hepatic hæmorrhages took place during the act of dying, and were due to the mode of death, *i. e.*, by suffocation; that, in fact, the extravasation was caused by the prolonged and violent action of the heart after the suspension of respiration.

CASE 2.—*Hemiplegia, left. Sudden in access. Frequent vomiting during next twelve hours. Sopor for three days. Sensation greatly diminished. Rapid formation of bedsores. Death in three and a half weeks.— Extensive hæmorrhage between extra-ventricular corpus .striatum and plane of white fibres separating it from insula. Mitral disease. Contracted granular kidneys.*

Mary B—, æt. 45, married and multiparous. Admitted into St. Mary's Hospital, February 2nd, 1871, died February 5th.

Last child six years old; catamenia regular, but diminishing. No previous illness, but liable to pain in the right side of the head and sickness at menstrual period.

On January 8th she had a slight attack of paralysis, but soon recovered.

On the 11th, *i. e.* three weeks before her admission, while at the house of a friend, she suddenly lost the use of her left limbs. There was no loss of consciousness at the time. She was taken home; vomited six or seven times in the course of the night; slept almost continuously for three days, waking at intervals, however, when she appeared to be quite sensible, and took food. The urine and fæces were passed in bed, and this continued to be the case after the patient recovered from her stupor. Bedsores formed rapidly, and a large anthrax came at the back of the neck where a blister had been applied.

On admission there were extensive sloughs on the left hip (the paralysed side) and over the sacrum; a large abscess in the right buttock and the anthrax mentioned above.

The left limbs were paralysed and relaxed; the mouth drawn to the right; not much deviation of the tongue. Sensation greatly impaired over right half of body generally (face, limbs, and trunk). The patient usually lay on her right side; the face and eyes turned to the right, but she was able to look to the left. She answered questions rationally, but wandered a good deal in the night. Pupils equal, of natural size, and acting well. No ophthalmoscopic examination (the ward lamp being out of order, and the patient dying before a second visit could be made).

Heart's action attended with thrill, perceptible to the hand below the left nipple; the thrill led up to the impulse. No murmur over any part of the heart. Apex beat below normal situation.

Legs slightly œdematous. No urine could be obtained for examination. Temperature 101·3°.

Post-mortem examination.—Brain. Nothing remarkable on surface of hemispheres; no embolism; no marked disease of arteries.

On section of hemispheres, just above level of corpus callosum, a blood-stain seen on right side to the outer side of the corpus striatum and thalamus. On further examination a large clot exposed, outer part brown, central portion black; walls of cavity ragged and soft. The cavity extended nearly the whole length of both ganglia, but scarcely reached the anterior end of the corpus striatum, and was between the corpus striatum externum and the plane of white fibres separating it from the convolutions of the island of Reil. The blood only invaded the ganglion at one point about the middle of its length, where a process of the clot penetrated the grey substance nearly up to the fibres running between the intra-ventricular and extra-ventricular portions of this body. Opening out the fissure of

Sylvius the thin lamina of nervous matter formed by the convolutions of the insula was easily torn through and the clot or cavity exposed. The thalamus was not involved, but was dissected off from the hemisphere equally with the corpus striatum.

The ventricles were empty, and apparently compressed by the clot which had displaced the corpus striatum and thalamus inwards.

Heart weighing 10¼ oz. Mitral valve contracted and thickened, and chordæ tendineæ shortened. The degree to which the orifice was narrowed not noted. Left auricle much dilated.

Kidneys small; much of cortical substance came off with capsule; surface coarsely granular and soft; a few cysts. Many purple points and patches, which on section looked like embolic infarctions. Cortex not greatly diminished.

Embolic patches in spleen.

Posterior zone of one eye removed. Apparently normal.

CASE 3. *Apparent drunkenness. Left hemiplegia coming on gradually with hemianæsthesia. Later unconsciousness. Hæmorrhage between corpus striatum and external capsule penetrating into lateral ventricle.*

William F—, æt. 60, cabman, admitted May 27th, 1875. Patient was brought in by police at 4 a.m. in an insensible condition. He had always been a steady man, but two years before had a pecuniary misfortune which preyed much on his mind.

Nine months ago he had a fit which lasted from 2 to 8 p.m. In this attack his wife says he was from the first unconscious (meaning probably delirious), but kept springing off the bed and rushing about the room, not knowing where he went, striking his head against the wall. He foamed at the mouth and bit his tongue, but did not speak until 9 p.m.; was considered epileptic by the practitioner who attended him. Ever since he has appeared " vacant," and complained of pain in the back of the head.

This was one of the "drunk or dying" cases about which sensational articles were written. He was noticed by a policeman about 1 a.m. sitting on the box of his cab apparently drunk. Was charged with being drunk and unfit to drive, and ordered to get down. He answered "all right" in a drunken sort of way, got down and into his own cab, in which he was driven to the police station. His wife, a most respectable-looking woman, said he did not drink.

The surgeon of the police called to him stated that he was brought in by the police between 1 and 2 a.m. He vomited, bringing up large quantities of undigested food smelling of liquor. He remained with him two hours, during which symptoms of paralysis of left side gradually came on. On which he sent him to the hospital.

Condition on admission.—Lies insensible, breathing noisily, respirations 32 per minute; occasionally making a sort of moan and tossing on the bed, using, however, only his right limbs. Lies rather on the left side, the limbs of which lie motionless. There is also a loss of sensation on the left side. If pinched on the right thigh he at once rubs the irritated spot, whereas he makes no such motion when pinched on the left side, though the spot be equally within reach of his right hand. There is considerable flexor rigidity of the left arm, and some rigidity of the leg; reflectorial movements may be produced by irritation of left limbs. Through his parted lips the tongue can be seen pushed very much to the left side. Both pupils react sluggishly to the light. Scarcely any perceptible difference between the two sides of the face. Eyes never move to left of orbit. Is able to swallow with difficulty. Vomited twice this morning.

Pulse rather weak, full between the beats. Temperature: left axilla, 100·8°; right axilla, 100·9° Urine, sp. gr. 1017, light-coloured, alkaline reaction, contains some albumen. This was drawn off with catheter: passes it under him. Five grains of calomel were given, an enema was administered, and an ice-bag was applied to the head.

May 28th.—Patient lies in much the same condition, but has as yet shown no signs of consciousness. Respiration 24; temperature 100·5°; pulse 80, irregular. Heart's action weak, no morbid sounds. Pupils much contracted. At 12.30 opened his eyes, and said, "The back of my head aches, so do not touch it." Lies in the same condition. Passes everything under him. Right extremities so restless that it became necessary to confine them, to prevent his throwing off the clothes. Respirations 44 per minute, less noisy; pulse 120, full, but weak and irregular; temperature 102·6°. Is perspiring very freely, much strong rigidity of left arm. Died at 5 p.m., no change having taken place up to that time.

Post-mortem examination, June 1st.—Encephalon. Considerable extravasation into meninges of posterior part of left hemisphere, and over back of both hemispheres of cerebellum. Bulging and flattening of convolutions of posterior part of right hemisphere. Membranes in both Sylvian fissures rather blood-stained, but no distinct extravasation. Bulging of island of Reil on right side, and slight extravasation into the surface of the right side of pons. The Sylvian and basilar arteries present evidence of not very extensive disease. A small aneurism on right vertebral cut across. Dura mater abnormally adherent to left hemisphere near longitudinal fissure. There was a very large clot in right lateral ventricle and its anterior and posterior horns, but not in the descending cornu. The blood reached the ventricle by an aperture which was at line of junction of the corpus striatum with hemisphere, dividing the body of the corpus striatum from the tail, and completely dissecting it away from the hemisphere. With this was a very large cavity in the hemisphere along outer side of ventricle, extending forwards nearly to the frontal extremity of hemisphere, and separated from the Sylvian fissure merely by the thickness of the gyri operti. The corpus striatum and thalamus were completely separated from the convolutions except by a small bridge close to descending horn. The clot in

addition penetrated into the corpus striatum, separating it from the thalamus along the line of the tænia semi-circularis. There was also some hæmorrhage into the lower part of the optic thalamus, and streaks of extra-vasation into the body of the corpus striatum. There was merely some bloody serum in the left ventricle. The left hemisphere and the cerebellum appeared quite healthy.

The heart was large, the left ventricle being very thick and of good colour; the valves healthy.

The liver was fatty and slightly congested.

The kidneys were small, contracted, and granular; cortex much wasted; capsule adherent.

The spleen appeared normal.

CASE 4.—*Giddiness and staggering; left hemiplegia; in-gravescent in access; loss of consciousness; vomiting. Hæmorrhage between corpus striatum and convolutions of insula.*

Mary R—, æt. 56, admitted August 5th, 1873. Died August 7th. Quite well up to time of attack; had been going about a good deal in underground railway. On leaving Edgware Road Station began to drop parcels she was carrying. Said she was giddy and staggered, but with the assistance of her daughter walked to John Street (quarter of a mile), where she sat down in a shop, and ten minutes later became insensible. Brought to the hospital an hour afterwards apparently quite insensible; left limbs paralysed and rigid; right arm constantly in convulsive motion; she opened her eyes when roused, but soon relapsed into coma; pupils equal and small; pulse full and bounding 80; respiration slow, left cheek puffed out in expiration; froth about mouth; tongue bitten on left side; vomited freely before admission.

Ice to head; sinapisms to calves; calomel gr. v; leeches to right temple; castor-oil enema.

August 6th, a.m.—Spoke distinctly this morning, told

her daughter she was in St. Mary's Hospital, having heard this over night when apparently unconscious.

2.30 p.m.—Apparently insensible; eyes closed; pupils small, sensitive to light; no distortion of face, but left cheek puffed out in expiration. Left arm relaxed and motionless; has seemed to move it a little. Left leg in similar condition; movement more decided; this leg rigid after arm had become relaxed; reflex action when soles tickled, more right than left. Swallows with difficulty. Heart sounds normal; pulse rather resistant; respiration very abdominal; inspiration sudden; expiration long (subject to bronchitis).

When roused answers very intelligently and clearly; no pain except at back of neck when moved; does not open eyes when requested; after speaking, pupils larger; urine and fæces passed in bed; temperature 7 p.m. 103·6°. She gradually became worse; much twitching of body; conjunctiva insensible; temperature 102·4°. Died.

Post-mortem examination.—Body remarkably stout and fat.

Brain.—Some congestion of surface and superficial bloodstains on left hemisphere. Right hemisphere softer to the touch. When about to open the fissure of Sylvius a rent appeared in anterior perforated space, exposing blood clot. This was found to extend far forwards into frontal lobe, and backwards along outer side of corpus striatum, between it and convolutions of insula, dissecting corpus striatum completely away from hemisphere, leaving it entire; ploughing up hemisphere to near thalamus and bursting into ventricle.

CASE 5.—*Hemiplegia with marked hemianæsthesia; ingravescent mode of attack; hæmorrhage between corpus striatum and thalamus and external capsule at posterior part of insula, with penetration of blood through thalamus into ventricle.*

Benjamin E——, æt. 64, a coachman, was admitted into

St. Mary's Hospital, December 11th, 1875, and died December 17th.

He was steady and sober; had enjoyed good health, but chalk stones were present in his fingers, and during the summer of 1875 he complained at times of pain in the back of the head.

He had not been sleeping well for a fortnight, but had been otherwise in his usual state of health, when, on the evening of December 10th, while grooming a horse, felt a numbness down the right arm, and experienced a peculiar general feeling which alarmed him. He found himself no longer able to use the brush, and sent his wife, who was holding the horse, for a medical man. He did not fall or lose consciousness, but on his wife's return could not walk upstairs, though he could still move the right arm barely. He answered the questions put to him by the medical man, but afterwards muttered to himself. At 8 p.m he vomited and gradually went off into a heavy doze, which continued till he was brought to the hospital.

On admission there was conjugate deviation of the head and eyes to the left; the head, however, could be turned to the right, but not the eyes; pupils equal; slight distortion of face; tongue carried to right when protruded; paralysis of the right limbs; very great impairment of sensation over entire right half of body and head. Not easily made to answer; speech indistinct and rapid, exactly like that of a drunken man; mind confused; did not know his wife. A systolic murmur was heard at apex of heart.

On the 13th, after a purging, he was less drowsy; answered rationally and distinctly, and talked about home affairs with his wife; pulse 64, high tension; slight inequalities in the course of the artery.

14th.—Says he is better and answers intelligently; is, however, unable to protrude the tongue, and failing to do this when asked tried to drag it out with his fingers. Lateral deviation of eyes still present; motions passed in bed; pulse 90; temperature 100·8°.

15th.—Answers "yes" to everything; unable to put out tongue, and uses fingers for the purpose; pulse 96; temperature now rising and much higher on the paralysed side; a.m., right axilla 103°; left 101·6°. 2.30 p.m., right 103·8°; left 102°. At 4 p.m. right 104°.

Ice-bags were applied to the scalp, and the temperature was reduced on the 16th to right 102·8°; left 101·4°; pulse 98. He got gradually worse, however, and on the 17th the pulse was 132; respiration 60; temperature, right 104·6°; left 103·2°. On this day he died.

Post-mortem examination.—Head,—dura mater firmly adherent to the skull; no thickening of either.

Arachnoid over top of hemispheres on left side thick and opaque. Much yellow subjacent fluid. Wasting of convolutions.

Arteries of base very atheromatous.

In right fissure of Sylvius much blood-stained fluid beneath membranes; gyri operti well marked.

In left, less fluid; posterior convolutions flattened out, and bulging. Clot not reached, however, by a slight incision. (*Anatomical note.*—A greater thickness outside proper wall of corpus striatum posteriorly, as fibres pass from temporo-sphenoidal wall of fissure of Sylvius to parietal wall beneath convolutions of island.)

On section, brain substance firm. In right lateral ventricle much blood-stained fluid; in left, clot and fluid. Section exposing left lateral ventricle also exposed the main clot, which lay outside posterior end of thalamus. Thalamus displaced inwards and forwards; intra-ventricular corpus striatum crowded forwards, its tail being turned outwards, so that the clot had pushed apart the two ganglia. The rupture into the ventricle had taken place at the inner side of the posterior part of the thalamus, and the effused blood had also penetrated between the posterior end of the thalamus and the *tegumentum*, tearing the thalamus and raising it up from the tegumentum. It had also carried away the anterior edge of the left superior corpus quadrigeminum.

The clot altogether appeared to be about the size of a walnut; it was flattened from above downwards. Anteriorly, the cavity from which it was dislodged did not pass forwards beyond the anterior end of the thalamus; its outer and lower wall was formed by the spread-out posterior convolutions of the island.

The hæmorrhage must have taken place much farther back than usual, and from the branch of the Sylvian artery described by Duret as passing to the thalamus, and the course of the blood inwards must have been determined by the process of the thalamus, which turns round the posterior edge of the crus. The lesion might have been described as involving the posterior part of the internal capsule, but this tract of fibres was little, if at all, damaged; a natural dissection had been performed by the blood raising the thalamus from the tegumentum.

The cases just related furnish sufficient evidence of the association of the ingravescent form of apoplectic attack with hæmorrhage of large amount between the lenticular nucleus and the external capsule. The occurrence of extensive extravasation of blood in this situation has already been noted by Charcot, Bouchard, Bourneville, Purvost, and Duret, and explained by the large size of the arteries here found. It is well known that the grey substance of the nerve-centres is much more freely supplied with blood than the white fibres, and that large vessels pass from the middle cerebral artery into the foramina of the anterior perforated space to the grey mass of the corpus striatum. Duret has shown by sections of injected brains that these arteries do not at once plunge into the grey substance of the ganglion and ramify in it, but run, some of them, one of large size in particular, round nearly its whole external periphery, between the grey matter and the external capsule—a fact which I had observed independently in the course of my dissections. Duret has also shown that they end in tufts and do not give off many branches like other arteries. It is obvious, as

pointed out by this observer, that in the large size of the vessels and in their mode of termination, we have an explanation of the liability to rupture and of the profuse haemorrhage. A further reason not previously taken into account why extravasation of blood in this situation is liable to be extensive is the absence of resistance. I have found that the plane of fibres forming the inner surface of the external capsule, though the fibres no doubt arise from the cell processes of the corpus striatum, is detached from the grey substance with extraordinary facility, so that the blood only performs a natural dissection in following the line of cleavage between them. In thus separating the capsule from the ganglion it will, moreover, probably cause rupture of other arteries running here.

Not only is there an absence of the obstacle to extravasation which would be offered by interlacing fibres, but the proximity of the fissure of Sylvius and of the descending cornu of the lateral ventricle diminishes the resistance to displacement and consequent pressure which solid brain substance would oppose. The Sylvian fissure is one of the provisions found in the brain for meeting variations in the vascular turgescence of the cerebral substance by corresponding variations in amount of meningeal fluid, and the room it affords for expansion of the island of Reil, can only be fully appreciated when the gyri operti of the latter are seen, as they are in these cases, not merely flattened but completely obliterated, so that the insula appears as a smooth, unconvoluted eminence. Further space is yielded by the compression of the descending cornu of the lateral ventricle, and by the displacement of the corpus striatum inwards.

The ready mobility of the parts surrounding the seat of haemorrhage, besides facilitating the extravasation, will also prevent any general or local pressure on brain substance till the mobility is exhausted, and to this I am disposed to attribute the late and gradual oncome of coma. The relation between pressure and coma, it is true, is a

question still in dispute, and it cannot here be adequately discussed. It is, however, generally admitted that the brain will bear without symptoms the gradual application of pressure, which, applied suddenly, would give rise to coma, and that sudden injury produces the unconsciousness of shock, or, as it was named by Trousseau, *cerebral surprise*, and it is clear that sudden pressure and forcible laceration are avoided in consequence of the anatomical facts here pointed out.

The extravasation is, however, rarely confined to the space between the extra-ventricular corpus striatum and its external capsule. When this is the case, the hemiplegia will probably be slight and temporary, and a minute account of the symptoms would be of extreme interest and value. It will easily be understood how the effusion of a considerable amount of blood before resistance is encountered favours the further progress of the extravasation ; since, according to the laws of fluid pressure, the distending force is the same at every point of the surface of the blood poured out as at the orifice of the bleeding vessel, and is therefore multiplied indefinitely. It will thus readily make its way, but always in the direction of least resistance; and here, as in all cases of cerebral hæmorrhage, the further course of the blood will be determined by the arrangement and relations of the fibres. When the ruptured vessel is at the anterior part of the corpus striatum, the blood travels forwards, and may penetrate into the frontal lobe; when on the lateral aspect the blood is directed upwards. The hæmorrhage never breaks through into the fissure of Sylvius, nor does the blood ever, so far as I know, enter and plough up the temporo-sphenoidal lobe; to do this it would have to penetrate continuous planes of fibres, which in the absence of antecedent softening never happens. At the upper angle of the extra-ventricular corpus striatum the plane of fibres which forms its proper external capsule, passing upwards, meets the mass of fibres of the corona radiata proceeding outwards, and an interlacement nearly at right angles takes place between them. At the line of intersection, too, the few arteries

which are sent from the middle cerebral to the intra-ventricular corpus striatum and to the centrum ovale of the hemisphere pass upwards and inwards, and also the far more numerous and larger veins conveying blood from the extra-ventricular corpus striatum and the mass of the hemisphere to the venæ Galeni. It might have been supposed that the intersection of fibres would oppose a barrier to the further extension of the hæmorrhage, but apparently interlacing fibres offer less resistance to rupture than a smooth continuous plane. No doubt, also, the vessels with the perivascular spaces afford a guide and open a way to the blood, since it almost invariably happens that the corona radiata is torn through partially or completely along the line of its emergence from the region of the central ganglia. The extravasation then either reaches the ventricle or ploughs up the hemisphere; in the latter case it may be directed into a longitudinal course in the substance of the parieto-frontal lobes by the mass of fibres of the great longitudinal commissural system, or may follow the central radiating fibres into the ascending parietal convolutions, which it splits up in the direction of their length.

It is the ingravescent mode of attack which constitutes the distinguishing character of the class of cases I have endeavoured to describe, and which I associate with hæmorrhage between the corpus striatum and external capsule. With the progress of the hæmorrhage new symptoms arise, which will differ according to the course taken by the blood. So far as the cases here related throw light on the symptoms attending different degrees and direction of further structural damage, it would appear that in all there were hemiplegia, hemianæsthesia, vomiting, and sopor. A noteworthy point is the very slight degree of facial hemiplegia observed, attributable perhaps to the distance of the lesion from the first or internal part of the lenticular corpus striatum, which is said to be specially concerned in the movements of the face and eyes. At the same time lateral deviation of the eyes was nearly always present. The patient in Case 2 survived for three weeks,

complete separation of the extra-ventricular corpus striatum and thalamus from the hemisphere. The rapid formation of bedsores indicated great impairment of the nutritive functions in the paralysed parts. In all the other cases the blood reached the ventricles, and a fatal termination occurred within a few days. In Case 1 laceration of brain fibres was attended with the symptoms enumerated and marked conjugate deviation of the head and eyes, while rupture into the ventricle caused profound coma and speedy death. In Cases 3 and 4 extensive ploughing up of the hemisphere with penetration into the ventricle at an indeterminate period gave rise to early rigidity of the paralysed parts, and constant movements of the limbs not paralysed. There was gradual loss of consciousness in both, but less profound and less durable in the latter, in which the blood took a direction forwards. In Case 5 the lesion was further back, and the injury to the thalamus greater than in any other case, and the impairment of sensation was very decided, while the loss of consciousness was not for some time so profound. The size of the clot was, however, much smaller.

It will be noticed that loss of sensation to a certain and indeed considerable degree has accompanied the hemiplegia, and this not only when the thalamus has been damaged, or when the posterior part of the internal capsule so called has been involved in the lesion, but when the hæmorrhage has been too far forwards for either occurrence. To this I beg to call special attention as hemianæsthesia has recently been too exclusively referred to lesion of the *posterior* part of the " internal capsule." There can be no doubt that injury of the fibres at this part will cause hemianæsthesia as observed by Charcot, Bourneville, and others, and shown experimentally by Carville and Duret and others. I desire simply to point out that impairment of sensation can be induced by lesion elsewhere, and especially in the situation I have spoken of outside the central ganglia and beyond the internal capsule.

This does not constitute a valid objection against the sensory function of the thalamus. The impairment of sensation is caused in the same way as the motor paralysis, which in these cases is due not to destruction of the corpus striatum, or to a lesion cutting it off from communication with the medulla and cord, but to separation of this ganglion from the hemisphere by rupture of the fibre of the corona radiata, and the same lesion which divides the communication of the corpus striatum with the hemisphere divides that of the thalamus. The fibres of the thalamus do not go merely or even mainly to the posterior part of the hemisphere, but pass forwards and outwards beneath the intra-ventricular corpus striatum to emerge from the region of the central ganglia in the corona radiata of which they everywhere form an important part. They are thus implicated when the corona is torn, and the cases show that separation of the thalamus from the hemisphere is attended with impairment of sensation as well as damage to the ganglion, or its separation from the sensory tract of the cord.

The object of this communication is to utilise the more extended and exact knowledge of the structural arrangements of the cerebral hemisphere in localising lesions occurring in different parts. It would have been more satisfactory to myself if I could have produced a parallel series of cases in which hæmorrhage had taken place in other situations than the one here considered, but opportunities for this have not fallen to me, and I have only the present contribution to offer to the notice of the Society.

DESCRIPTION OF PLATE XIV.

Figs. 1 and 2.—Transverse section of brain. Fig. 1, at anterior perforated space, and Fig. 2, in front of optic commissure, showing (Fig. 1) origin, and (Fig. 2) further distribution of arteries of corpus striatum. After Duret.

C C, corpus callosum; S v, ventricular corpus striatum (nucleus caudatus); Sx, extra-ventricular corpus striatum (nucleus lenticularis); r c R, fibres entering below r c from crus cerebri, emerging above R as corona radiata, constituting so-called internal capsule; F S, fissure of Sylvius; I R, island of Reil; c, thin layer of grey matter, claustrum, between which and grey substance of corpus striatum is thin plane of proper white fibres of corpus striatum called external capsule.

Fig. 3.—Vertical section passing obliquely from crus cerebri to fissure of Sylvius on outer aspect of hemisphere. Th, thalamus.

The representation of fibres is diagrammatic.

Plate XIV

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Fig. 2.

Fig 1.

F i g 3

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ON THE

CAUSATION OF THE WATER-HAMMER PULSE,

AND

ITS TRANSFORMATION IN DIFFERENT ARTERIES AS ILLUSTRATED BY THE GRAPHIC METHOD.

BY

A. L. GALABIN, M.A., M.D.,

LATE FELLOW OF TRINITY COLLEGE, CAMBRIDGE; ASSISTANT OBSTETRIC
PHYSICIAN TO GUY'S HOSPITAL; AND ASSISTANT PHYSICIAN
TO THE HOSPITAL FOR SICK CHILDREN.

THE characteristic pulse of aortic regurgitation is so familiar to the observation of every one that it may perhaps be thought that its mode of origin must be so well known that there can be little room for further discussion on the subject. With regard to the theory of its causation, little or nothing has, I believe, been added to the explanation given by Sir Dominic Corrigan in his first well-known paper. This paper, published in the 'Edinburgh Medical Journal' in 1832, gives indeed an account of most of the characters which are now recognised in this form of pulse. Sir Dominic Corrigan there describes, as a symptom of the permanent patency of the aortic orifice, a visible and forcible pulsation of the

arteries, most distinct in those of the head and neck, especially the carotids and temporals. He states that it is there most visible in the erect posture, and that it becomes more distinct in the radial artery when the arm is raised. He explains it as due to the fact that the aorta pours back blood into the left ventricle, and becomes flaccid during diastole, the result of which is that the arteries adjoining become also rapidly emptied. The fact that the character is most marked in the arteries of the head and neck he ascribes to the circumstance that these most readily empty themselves backward, being assisted by the gravity of the blood. The same reason he applies also to account for the effect produced by elevation of the arm. He considers that the strength of the heart has but little to with the result, since the pulsation is but little marked in the lower extremities. I believe that the explanation thus given by Sir Dominic Corrigan is that which is now generally accepted, except that most observers would now consider the dilatation and hypertrophy of the left ventricle to be a more important element in the matter. We may consider, then, that the received explanation of the pulse of aortic regurgitation is, that it is simply the effect of an ample pulse-wave being thrown into empty arteries, and that the reason why it is developed by elevating the arm is solely that the radial artery is thus rendered more empty during diastole.

In the present communication I propose to consider whether all the characters of the aortic pulse, as discovered by the finger and by the sphygmograph, are fully accounted for by this theory, which in itself is undoubtedly a true one, or whether any further mechanical explanation is required or can be discovered. I will first refer to a character on which Sir Dominic Corrigan does not lay stress in the article which I have quoted, but one which is well known to every one, and which has given to this form of pulse the title which I have taken in the heading of my paper, namely, that of water-hammer

pulse. The quality which is well expressed by this name is that of extreme suddenness in the commencement of the pulse, which in marked cases gives to the finger the impression of a sudden blow or jar. I think it is not too much to say that this is *the* diagnostic quality of the pulse of aortic regurgitation, for a merely ample wave entering a relaxed artery may be found, to a considerable extent, under other conditions, as for instance in persons who have habitually relaxed arteries, when under the influence of excitement, or violent muscular exercise. Moreover, skilled observers do not hesitate to found a diagnosis of aortic regurgitation on this sole quality of suddenness in some instances where the pulse-wave is not ample at all, as in cases where mitral is combined with aortic regurgitation. It may be added that it is the quality of suddenness which is especially developed by the elevation of the arm. Now, in the explanation which I have mentioned there is nothing to account for this suddenness, as apart from the mere height and fulness of the pulse-wave. For we know, as a general rule, that a hypertrophied muscle commences its contraction in a more slow and deliberate manner than one which is weak. This is confirmed in the case of the heart by the dull and prolonged first sound associated with hypertrophy, as opposed to the short and sharp first sound of dilatation. The point can also be tested by direct measurements. From cardiographic tracings I have found that the interval occupied by the main upstroke of the trace, which indicates the time which the ventricles take in becoming fully hardened, may be increased in cases of aortic disease from its normal value of about one twelfth of a second to as much as from one eighth to one sixth of a second, that is to say, that it may be nearly doubled. There is therefore no reason to think that the hypertrophied heart of aortic disease would throw a more sudden wave than usual into the aorta, and I need not say that the water-hammer pulse is not confined to those cases, if such there be, in which there is dilatation without hypertrophy.

There is another quality about the aortic pulse which I would mention from my own observation, namely, that it is less marked in the large arteries than in those which are somewhat smaller, as the temporal, facial, or radial, which are often seen pulsating much more violently in proportion. I believe, indeed, that the quality of suddenness is scarcely marked at all in the largest arteries, the carotid, subclavian, or abdominal aorta. I hardly know whether this is generally recognised as being the fact, but I will refer hereafter to the evidence on this point afforded by the sphygmograph.

Now, the quality of pulse which depends simply upon an ample wave being thrown into an empty artery would of course be more marked near to the heart than at a distance, since the aorta would more readily empty itself backward than the more distant arteries.

I would say, then, that *there are two qualities of the aortic pulse manifest to ordinary observation, one of the two being its most diagnostic quality, as to which the common theory of its causation offers no explanation whatever.*

I pass on to the evidence afforded by the sphygmograph as to the aortic pulse. As might be anticipated, one of its characteristics, in marked cases, is its extreme amplitude when taken at a low pressure. This, when it is present, is sufficient to be diagnostic, but it is absent in milder cases. An instance of it, although by no means so extreme as may often be found, is shown in Pl. XV, fig. 11. This was taken from a patient in whom only a systolic murmur at the base and no diastolic murmur could at the time be detected, although the pulse, both to the finger and the sphygmograph, had a marked quality of regurgitation. One of the most important and characteristic
is that
be seen
11, 12,
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aortic pulse is the emptiness of the artery during the diastolic period. In many cases, however, when the pulse has to the finger the aortic quality, this flatness does not appear in the trace, and probably many persons have on this account been disappointed at finding less assistance from the sphygmograph than they expected in the diagnosis of aortic regurgitation.

In Pl. XV, fig. 12, is shown the trace of a pulse which to the finger had a distinct, although by no means an extreme, aortic quality. In this there is no flatness whatever in the diastolic portion, and the only thing abnormal about it is its great amptitude and the suddenness of the upstroke. This latter quality is not obvious to the eye, and is only adequately appreciated when arcs of circles are drawn through the different points of the curve, and accurate measurements are taken. Thus, the general shape of the curve in Pl. XV, fig. 12, does not seem very different from that of the healthy pulse shown in Pl. XV, fig. 2, but on taking measurements it is found that the upstroke of the aortic pulse (fig. 12), although higher, occupies about one third less time than that of the healthy pulse in fig. 2. I believe that in all instances in which the flat diastolic portion is seen in traces taken at a somewhat high pressure it would be replaced by a descending line in the same situation if the trace were taken at a lower pressure, but this the dimensions of the recording plate will frequently not allow, so great is the amplitude of the pulse when pressure is very low.

Now, it is to be remembered that a similar flatness in the diastolic part may be produced in a healthy pulse by applying too strong a pressure. Nevertheless, the fact that such flatness is readily produced by a pressure much less .... is of great diagnostic .... rather as an indica- .... as a delicate sign of .... den emptying of the .... it would be seen in
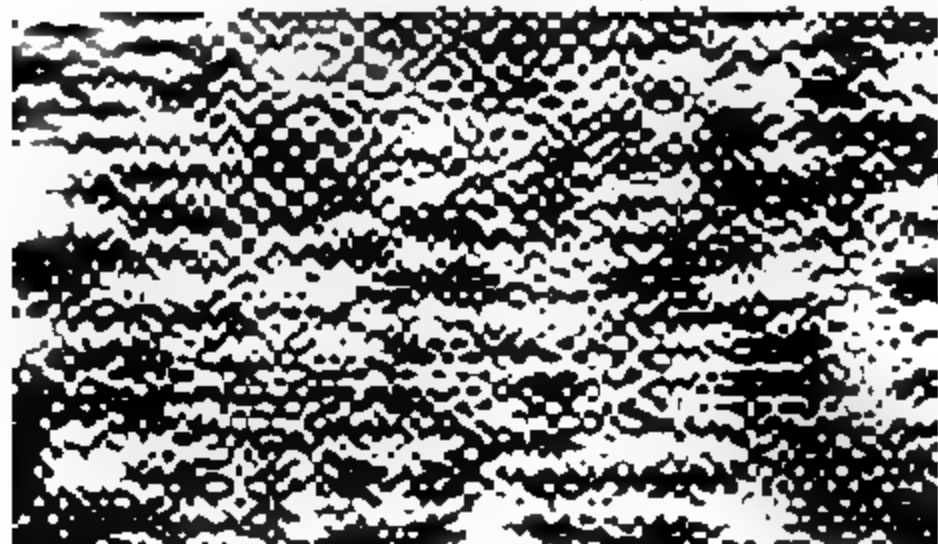
a trace taken at a lower pressure, but merely that the tension throughout the diastolic period is *greatly* less than that during systole.

In Pl. XV, figs. 4 and 5, taken from a case of extremely free regurgitation, is shown the flatness readily brought out by a moderately increased pressure, namely, one of 4½ ounces (*vide* fig. 5), although the most perfect trace (fig. 4), taken at a pressure of three ounces, which is about the pressure applicable to a normal pulse, shows no flatness whatever.

Closely allied to the flatness in the diastolic portion is the absence, or relative smallness, of the dicrotic wave. I shall avoid entering here on the vexed question of the causation of dicrotism which I have elsewhere discussed. I will remark only that great expectations might be raised as to the diagnostic value of the trace from the common view that the dicrotic wave is due solely to the closing of the aortic valves, for then it should be absent or much diminished in cases of regurgitation, but that these expectations will be in great measure disappointed. Other things being equal, the dicrotic wave is less when the aortic valves are imperfect, but the degree of dicrotism is affected by many other conditions besides the state of the valves, and it may appear to be as great as, or sometimes even greater than, normal in a pulse which has to the finger a distinct aortic quality, as will be seen in Pl. XV, figs. 11 and 12, where the dicrotic wave is indicated by the letter c. A considerable diminution or absence of the dicrotic wave is, however, a valuable sign of freedom of regurgitation.

It is interesting to note the effect of fever on the pulse of free aortic regurgitation, an instance of which is given in Pl. XV, fig. 17, taken from a patient suffering from pneumonia, whose temperature was 103·1°. The pulse has become hyperdicrotic, but the actual magnitude of the dicrotic wave (c) in proportion to the primary is much less than it might be expected to be with this degree of fever. Moreover, probably in consequence of

the cardiac hypertrophy, there is still seen in the trace the tidal wave (B), which is generally lost at such a temperature, and before the pulse becomes hyperdicrotic. When the aortic quality of the pulse is more extreme, even a great elevation of temperature may fail to bring out any dicrotism, and if there is very great hypertrophy, there may be still a marked tidal wave, even when the pulse is very rapid, indicating a comparatively prolonged systole, in place of the short contractions of the ventricle, which generally occur when the pulse-rate is much increased. Both these qualities are strongly marked in the pulse shown in Pl. XVI, fig. 12, which was taken from a patient suffering from extreme aortic regurgitation, complicated by pneumonia. He had at the time a temperature of 104° and pulse of 126, and he died within twenty-four hours from the time when the tracing was taken. The tidal wave is marked in the figure by the letter B, and no dicrotic wave is visible.

The abnormal development of the tidal wave, which, in the instance just mentioned, persisted even at a temperature of 104°, is a very general characteristic of aortic pulses in the absence of fever. In these cases it has not quite the same interpretation as when the vascular system is healthy.

A pronounced tidal wave has become commonly known as a sign of increased arterial tension, understanding by this term not merely an increase of the tension reached at the summit of the wave, but an elevation of the minimum tension at the end of diastole, in consequence of a diminished freedom of outflow from the arteries. This interpretation is established by the experience which shows that such a tidal wave, the vascular system being healthy, is associated, as a rule, with an increase, not only of the pressure required to extinguish the pulse, but of that which it will bear without diminution, or much diminution of the amplitude of the trace. It is confirmed by the consideration that the systole of the heart will be prolonged if it meets with unusual resistance, owing to an obstructed

outflow from the arteries. The evidence of elevated ten-
sion derived from the tidal wave should, however, always
be corroborated by that afforded by the recorded pressure,
and I think that it has been used by some who have com-
paratively small experience in sphygmography, without a
sufficient regard for the necessary qualifications. That it
does not always hold good in cases of valvular disease is
shown by the fact that the tidal wave attains its very
greatest development in cases of great obstruction of the
aortic orifice (Pl. XVI, fig. 8), a condition which certainly
does not tend to increase arterial tension. Apart from any
change which the pulse-wave has undergone in receeding
from the heart, a marked tidal wave directly indicates a
prolongation of ventricular systole.

Now, in most cases of marked aortic regurgitation in
which there is a large tidal wave, it is found that the
pressure required to extinguish the pulse is much in-
creased, but that the greatest amplitude of curve is
obtained at a low pressure, and that even a moderate
increase of pressure is often sufficient to flatten its dias-
tolic portion. This confirms the conclusion, which is
almost self evident, that in aortic regurgitation the mini-
mum arterial tension is rather diminished than increased,
the arteries being able to empty themselves backwards as
well as forward. The prolonged systole in such cases is
therefore simply due to the hypertrophy of the heart.
Hence a distinction must be made between the increase of
maximum and that of minimum arterial tension ; and it
should be remembered that the former only, and not the
latter, is found in aortic regurgitation.

I have next to consider the evidence afforded by the
sphygmograph as to the effect produced upon the radial
pulse by the elevation of the arm. By this means the
water-hammer quality of the pulse is often developed in
an extraordinary degree, and the finger sometimes receives
the impression that the force of the stroke against it is
actually increased. In many cases, however, this is not
confirmed by the sphygmograph, as may be seen by a

comparison of figures 3 and 13 in Pl. XVI, taken while the arm was elevated, with the corresponding traces, figures 2 and 12, taken when it was horizontal. The height of the upstroke is generally diminished, as well as the pressure which the pulse will bear, and it would seem that the wave loses somewhat of its force by ascending in opposition to gravity. The eye does not detect so readily as the finger what is the peculiar quality about these pulses. But if arcs be drawn through the several points, and measurements be taken, it is found that the suddenness of the upstroke is increased. In the two instances given the time occupied by the upstroke, with the arm elevated, is only about two thirds of the corresponding interval, already shorter than normal, with the arm horizontal. It is therefore, in many cases, solely the quality of suddenness, and not that of fulness in the aortic pulse, which is increased by elevation of the arm. Probably, indeed, the suddenness thus produced is not quite adequately represented in the sphygmographic trace, since suddenness of motion is of all qualities the most difficult to register by any recording instrument, the mechanism of which is apt to lag behind the motion to be followed, when it is of a very abrupt kind.

In some instances, on the other hand, the amplitude of the trace is actually increased by elevation of the arm. Usually the pressure applied to the artery must be diminished to obtain this result, but the fact remains that, in these cases, the most ample trace with the arm raised is loftier than the most ample curve which can be procured when it is horizontal. Occasionally the same result is found even when the pressure applied is the same in the two cases, an instance of which is shown in Pl. XVI, figs. 14 and 15, the former taken with the arm horizontal, the latter with the arm raised, and both at a pressure of three ounces. The case was one in which the water-hammer quality of the pulse only became manifest upon elevation of the arm. I think that a comparison of the curves will show that these require some further explanation than

that afforded by the common theory of the aortic pulse. For according to that theory the increased amplitude is explained on the ground that, when the arm is raised, the artery is more completely emptied during diastole, and thus the base-line of the curve is lowered, so that its summit appears more lofty. This, however, is not sufficient to account for the difference in these curves. For, in the first place, the flatness of the diastolic portion of the curve in figure 14, in which the aortic notch descends almost to the level of the base-line, shows that the artery was already, when the arm was horizontal, almost completely flattened under the spring of the sphygmograph even at the beginning of that portion of the wave which corresponds to diastole; and secondly, the systolic wave in figure 15 has not simply acquired an increased elevation above the rest of the curve, but its shape is transformed, the primary summit having become more lofty and pointed, and the tidal wave lower than that in figure 14. In other words, the wave has undergone an increased degree of that change, which, as will hereafter be shown, always takes place in the aortic pulse as it recedes from the heart.

It is not very easy to obtain reliable tracings for comparison when the arm is raised, and some precautions are necessary to avoid error. For, in general, it is better to dispense with the small secondary spring, used to keep the recording lever in apposition with the knife edge of the sphygmograph, but when the arm is raised this must be employed, since the lever is no longer sufficiently kept down by its own gravity. The secondary spring therefore should be so delicate as only just to prevent the lever from separating from the knife edge, and tracings, with the arm horizontal, should first be taken both without and with the small spring, in order to observe the change, if any, produced by its presence, and ensure that the difference so made is unimportant.

I think that a careful estimation of the quality developed in the aortic pulse by elevation of the arm, even as

ascertained by the finger only, will be in agreement with the conclusion that it belongs to the upstroke of the wave only, and that, in most cases, it consists chiefly of the character of suddenness. For since the finger scarcely detects the artery during the diastolic period, except when abnormally thickened, its greater or less emptiness at that time can have but little effect on the impression conveyed; and I believe that most observers, on analysing the process of judgment, will find that they draw their conclusion as to an aortic quality solely from the stroke against the finger, without waiting for the diastolic portion of the wave.

I pass on to the changes of the aortic pulse in the different arteries, as revealed by the sphygmograph; and I will first draw attention to a case in which these changes were unusually marked. In Pl. XVI, figs. 9, 10, and 11, are shown tracings from the carotid, radial, and dorsalis pedis arteries of a patient suffering from aortic regurgitation, which were taken while he was lying in bed. It will be seen that, in the carotid artery (fig. 9), the systolic portion is extremely broad and square, corresponding to the prolonged systole of the hypertrophied ventricle. In the radial artery (fig. 10) the systolic portion is still broad, but the diastolic portion has become more flattened, while in the dorsalis pedis (fig. 11) the pulse-wave has become completely transformed; the primary wave is exceedingly sudden, lofty, and narrow, and is followed by a long flat interval. The characters of the pulse to the finger corresponded exactly to that seen in the tracings, for in the carotid artery there was no perceptible suddenness, in the radial it was somewhat marked, but in the dorsalis pedis, an artery whose pulsation is not generally very distinct, the water-hammer quality was developed in the most extreme degree possible. In all other instances which I have examined by the sphygmograph the sudden, lofty, and pointed primary wave has been absent in the arteries nearest to the heart, and has only been developed at a certain distance from it.

A series of tracings corresponding to the last is shown in Pl. XVI, figs. 5, 6, and 7. These were taken from the subclavian, radial, and dorsalis pedis arteries of a patient who was suffering from aortic regurgitation of a very free character, as is indicated by the almost entire absence of the dicrotic wave in the traces. In the subclavian trace (fig. 5) the primary upstroke is even more gradual than is usual in the normal pulse, and there is no flatness in the diastolic portion. In the radial pulse (fig. 6) the systolic wave has become relatively higher and steeper, but it is still broad at the summit; in the dorsalis pedis (fig. 7) it has become much more pointed, while its right hand shoulder, formed by the tidal wave, has become slanted off. A corresponding change is manifest in the singular series of tracings shown in Pl. XVI, figs. 1, 2, and 4, also taken from a case of free aortic regurgitation. In the carotid artery (fig. 1) the second summit of the bifurcated systolic wave is higher than the first. In the radial pulse (fig. 2) the first summit has become the highest, and in the dorsalis pedis (fig. 4) the first summit has become very high and lofty in proportion to the rest of the trace, while the second summit (B), which is formed by the tidal or predicrotic wave, is very slightly marked in comparison, although it retains about the same relative position. Yet another series of a similar kind is shown in Pl. XV, figs. 6, 7, and 8, taken from the radial, femoral, and dorsalis pedis arteries of another patient suffering from aortic regurgitation. The femoral trace does not differ from that of the radial or dorsalis pedis so widely as does the curve taken from the carotid or subclavian; but these tracings also show exactly the same kind of change. The systolic wave is much more lofty, narrow, and sudden in the dorsalis pedis trace (fig. 6), while the dicrotic wave (c), which in the radial was well marked, has become almost entirely lost.

I think that the evidence afforded by these tracings, which in all cases were in accordance with the quality of the several pulses as manifest to the finger, is sufficient to

show that *the water-hammer quality of the aortic pulse does not exist in the pulse-wave as it is thrown into the aorta, but is a character which is gradually developed as the wave recedes from the heart, and reaches its maximum at a point which, in some cases, is as distant as the dorsalis pedis artery.*

It is true that the carotid arteries are often seen in these cases to pulsate strongly, but this is merely the effect of the amplitude of the wave, due to the dilatation and hypertrophy of the left ventricle, and not to its suddenness. It is perhaps unusual for the water-hammer quality to be so relatively prominent in the dorsalis pedis artery as it was in the cases of which I have shown tracings. I have observed that this happened when this quality was not very marked in the radial artery, and the maximum of suddenness seemed only to be acquired at a greater distance from the heart. When the radial pulse is extremely sudden, that of the dorsalis pedis is generally but little so, for the maximum of suddenness has then been reached at a shorter distance from the heart, and the wave is afterwards rendered progressively more gradual, a change which the elastic walls of any tube tend to produce in all waves which pass through it. I think, however, that it is important to remember, as a practical aid to diagnosis, that in a few cases in which the aortic quality is absent, or but slightly marked in the radial pulse, it may be distinct in the dorsalis pedis, when the leg is horizontal, or, still better, when it is elevated.

In order to arrive at a correct understanding of the cause of this acquired suddenness of the aortic pulse, it is necessary to compare the changes which it undergoes with those of various forms of pulse, when no valvular disease is present. This physiological change in the pulse-wave I have discussed in a paper published in the ' Journal of Anatomy and Physiology' for January, 1876, founded on a large number of tracings taken from the different arteries of the body, and from different points of

a schema representing the arterial system, made up of bifurcating elastic tubes, and adapted to an artificial heart of india rubber. I found that in all cases the systolic wave underwent a progressive change of such a kind that its right hand portion, generally made up of the tidal wave, became gradually shaved off, while the primary summit became narrower and comparatively loftier than before. At the same time the descending curve of the wave became more and more gradual, as all waves transmitted through elastic tubes eventually do, but this change did not occur in anything like a corresponding degree in its ascending curve, but on the contrary this often retained its steepness for a considerable distance. There was therefore evidence of the existence of some cause which, with regard to the ascending curve of the wave, counteracted, up to a certain point, the tendency which the elastic walls of tubes have to render gradual, and eventually obliterate, all waves. The change of shape of the systolic wave may be described as a progressive piling up of more and more of its volume in the front of the wave, that is to say, at its left hand side, as it appears in the trace, while its descending portion acquires a more gradual slope. This change as seen in the healthy pulse is shown in Pl. XV, figs. 1, 2, and 3, taken from the carotid, radial, and dorsalis pedis arteries of a healthy man, who had a somewhat unusually strong and full pulse. In Pl. XV, figs. 9 and 10, are shown tracings from the tubes of the schema which have a considerable resemblance to the human pulse, and show all the secondary waves which are seen in it. Fig. 9 was taken from the main trunk of the schema, not far from the aortic valves, and fig. 10 from a small tube at a distance of four and a half feet from the former. It will be seen that the systolic wave in the latter curve has undergone a similar change to that which occurs in arteries.

There are some cases, even when there is no valvular disease, in which the pulse-wave undergoes a far greater

transformation than that which normally occurs. In Pl. XV, figs. 13, 14, and 15, are shown the carotid, radial, and dorsalis pedis arteries of a patient suffering from phthisis. His arteries were exceedingly relaxed, and he had at the time a temperature of 102·8°. It was noticeable also that there was a marked interval between the pulses in the different arteries, indicating an unusually slow transmission of the pulse-wave. It will be seen that in the carotid pulse, fig. 13, there is a marked tidal wave (B), which forms the summit of the trace, and dicrotism is not great. In the radial artery, fig. 14, the systolic wave has undergone the usual kind of change, but in a very unusual degree. The primary summit has become narrow and pointed, and the tidal wave is almost lost. In this instance another curious change has also taken place : the pulse has become hyperdicrotic, and the dicrotic wave of considerable magnitude as compared with the primary. In the dorsalis pedis artery, on the other hand, the primary wave is still pointed, although less so than in the radial, but the dicrotic wave is entirely lost, and its former place is occupied by a flat interval. The unusual degree of transformation in the pulse-wave which took place in this patient is not usually found in febrile or dicrotic pulses, and the only thing which could be found in him to account for it was the extremely lax condition of his arterial system.

The result, then, of the comparison of the several series of pulses in aortic regurgitation with similar series taken from persons having healthy vascular systems is to show that *the transformation which the systolic portion of the pulse of aortic regurgitation undergoes, and by which the quality of suddenness is developed, is only an exaggeration to an extreme degree of a change which takes place to some extent in the normal pulse.*

In seeking the mechanical explanation of this change of shape we are naturally led to look for analogy to the modifications which other kinds of waves undergo. Now, a precisely similar change of form is undergone by waves

on the sea, or on the surface of any water, as they approach the shore. The wave becomes more lofty, and its front progressively steeper, while its descending curve grows more gradual, until at length the front becomes so steep that the wave curls over and breaks. The mechanical explanation of this is well known. It depends upon the principle that each individual portion of a wave may be regarded as itself moving as a separate wave, the effects of which are combined with those of all similar component waves. The whole wave-motion is then the aggregate formed by adding together the motions due to all the parts, or component waves, into which it is divided. If, then, the conditions of motion are different at different parts of the wave, the velocity of transmission may be also different at different parts, and, if this is the case, the shape of the wave will become altered as it proceeds. Now, in the case of a wave on the surface of water, we know from hydrodynamics that the wave-velocity is greater the greater the depth of the water. The depth of the water is of course greater at the summit of the wave than at its base, and hence the crest of the wave has a greater velocity than its front, and constantly tends to overtake it. So long as the water is deep this effect is extremely slight, and is overcome by the tendency which all waves have to become more gradual as they proceed. As soon, however, as the water becomes very shallow, the difference between the depth at the summit and that at the base of the wave becomes considerable in proportion to the whole depth. The crest of the wave then rapidly overtakes its commencement, so that the front of the wave grows steeper and steeper, until at length it breaks.

A precisely similar cause acts, under some circumstances, upon another sort of wave, extremely different in shape and dimensions, namely, the tidal wave. When concentrated by funnel-shaped estuaries, this sometimes acquires, in consequence of the shallowness of the water, the steep and broken front which is known in some rivers

by the name of the "bore." The bore on the Severn or the Ganges has thus a precisely analogous cause to the breaking of the smallest ripple upon the bank of a pond.

In order, then, to find an explanation in the case of pulse-waves, we have to examine the velocity of transmission of waves in elastic tubes. The mathematical solution of this question is one of great difficulty, for it requires as a starting-point a knowledge of the laws, to be ascertained by experiment, which connect the tension and the expansion of elastic tubes, and these have not been sufficiently ascertained. I have not therefore been able to arrive at an accurate calculation of the actual velocity, but I have succeeded in deducing the conclusion, that the velocity is greater the greater the pressure within the tube. Since, however, there is something doubtful in the data for calculation, it is fortunate that this result can be easily confirmed by experiment, and my observations have shown me that this law does hold good. More elaborate experiments on the point have been made by Dr. Marey, and are described in his recent work ('Physiologie Expérimentale,' Paris, 1875). He has estimated the wave-velocity by taking a very large number of simultaneous tracings from successive points of the tubes under observation, and he arrives at the same conclusion, namely, that the velocity is greater the greater the tension (op. cit., p. 111). Thus he finds that, if a succession of waves be thrown into the tube, the velocity of transmission progressively increases as the tension rises (op. cit., p. 115). The same explanation will apply to another result which he observes, namely, that the velocity of each wave progressively diminishes as it travels onwards; for as the wave becomes flattened as it proceeds, the tension at its summit will become less, and its velocity will be diminished in proportion.

Now, as the pulse-wave proceeds along an artery it is manifest that the tension at the summit of the wave is much greater than that at its base. The summit of the wave will therefore have a greater velocity than its front,

and will constantly tend to overtake it.   The result will
be that the highest point of the wave will tend to become
nearer and nearer to its commencement, and more and
more of its volume to be piled up at its front, so that the
front itself becomes steeper.   This is precisely the kind of
change which the tracings show actually to take place in
the pulse-waves.   In the normal pulse, however, this ten-
dency, as far as regards the steepness of the upstroke,
seems to be pretty much counteracted by the opposing
influence, namely, the tendency of the elastic arterial walls
to render all waves more gradual, and eventually to
obliterate them entirely.   The influence tending to make
the upstroke steeper is proportional to the difference of
tension between the summit and the base of the wave.
This will obviously be very much greater in cases of
aortic regurgitation, in which a powerful wave is thrown
into the vessels, and the arteries become very empty
during diastole.   We may expect then the result which
we actually find, that in such cases the influence tending
to make the front of the wave steeper preponderates, and
the quality of suddenness in the pulse is developed as it
recedes from the heart, so that the well-known water-
hammer character is thus produced.   This effect, however,
must at some point reach a maximum, after which the
obliterating influence alone is at work, and both upstroke
and downstroke are gradually rendered slanting, somewhat
as the wave approaching the shore loses all its steepness
when once it has broken.   We must expect, then, that, if
the pulse of aortic regurgitation has become very sudden
at a moderate distance from the heart, as in the radial
artery, it will have lost its suddenness at a greater distance,
as in the dorsalis pedis.

The same principle fully explains also why the quality of
suddenness is increased by elevation of the arm or leg.
For the arteries are then rendered more empty during
diastole, the difference between the tension at the summit
and that at the base of the wave is increased, and there-
fore the tendency to render the front of the wave steeper,

which is proportional to this difference, is increased like-wise. It may perhaps be regarded as a confirmation for this explanation, that the case in which a great trans-formation in the pulse-wave was found apart from any valvular disease (Pl. XV, figs. 13, 14, and 15), was one in which the arteries were excessively lax, and therefore the tension very low during diastole.

It may perhaps be thought by some improbable that there should be any analogy between waves transmitted through an elastic tube and waves on the surface of water, which seem to be very different in their nature. It is well known, however, to mathematicians that there is a certain degree of analogy in all kinds of wave motion, and close correspondence sometimes exists between waves produced under very different circumstances. Thus the same form of equation is found to represent the motion of waves on the surface of a straight closed trough or canal of water, and also the transverse vibrations of elastic strings, and the peculiarities of the oscillations have a close resemblance in the two cases, although they are very slow in the one and very rapid in the other. With regard to pulse-waves and waves on the surface of water, the similarity in the change of shape results from a resemblance in one point only, namely, that in both there is a cause which renders the velocity of transmission greater at the summit than at the base of the wave, that cause itself being quite different in the two cases. The only other resemblance in the nature of the two motions necessary to render the analogy valid is the undoubted fact that each is transmitted as a wave.

For the production of a change of the shape of the wave in the manner which I have described, it is of no consequence whether the motion of the particles of fluid is transverse to the direction of propagation of the wave, or longitudinal, or whether, as is the case in most waves, and in both of the forms of wave under consideration, the motion of each particle is partly longitudinal and partly transverse. The wave on the surface of water is convenient for the pur-

pose of illustration, because a considerable proportion of the motion of the particles is transverse, and the wave has therefore a visible shape.   A similar transformation might, however, take place even in a wave in which the motion of the particles of fluid is entirely longitudinal, such as that produced by the propulsion of an elastic fluid through a non-distensible tube.   The only difference is that in such a case the wave would not be visible, unless rendered so by some special contrivance, and therefore the change of its shape would not be manifest.   In the case of the pulse there is some lateral as well as a longitudinal movement of particles, and a true wave of lateral expansion in the artery occurs.   It is true that the sphygmographic trace indicates directly the wave of variation of pressure in the artery rather than the wave of expansion, for upon the pressure within the artery depends the degree of flattening which it undergoes under the spring of the sphygmograph.   Since, however, the expansion of the artery when uncompressed varies with the pressure within it, although not according to a strict arithmetical proportion, one quantity may be taken as the representative of the other, and the sphygmographic curve will nearly express the shape of the magnified wave of expansion of the uncompressed artery.   It is scarcely necessary to add that there can be nothing in the case of arteries which corresponds to the breaking of a wave on the surface of water, except the fact that in the former case also there may be a certain point at which the front of the wave reaches its maximum of steepness, after which it becomes progressively more gradual.

It is instructive to observe the contrast in the change of shape of the dicrotic as compared with that of the primary wave.   In all cases the ascending curve of the dicrotic wave (c) begins from the first to become rapidly more gradual, instead of, like the primary wave, retaining its steepness for a considerable distance.   This will be seen best by a comparison of the tracings taken from the carotid, radial, and dorsalis pedis arteries of a healthy person, shown in Pl. XV,

figs. 1, 2, and 3. The distance from the commencement to the summit of the dicrotic wave in the radial artery is about three times as great as in the carotid, and in the dorsalis pedis about four and a half times as great. The same change is apparent in numerous corresponding traces obtained by Wolff from the radial and dorsalis pedis arteries, and also in some published by Professor A. H. Garrod. The explanation is that the difference in tension between the base and summit of the dicrotic wave is but slight, the tendency to make the upstroke steeper is therefore slight likewise, and is far overpowered, even from the commencement, by the obliterating influence of the elastic arterial walls, which tends to make both upstroke and downstroke alike more gradual, and the base of the wave broader.

As seen in the tracing of the aorta, which may sometimes be obtained when that vessel has undergone aneurismal dilatation, without the formation of any sacculated aneurism, the ascent of the dicrotic wave is still more abrupt and steep than in the carotid. So different does it look in this form from the appearance which it has in the radial pulse that Marey, who obtained the abrupt pointed wave in his tracings representing the pressure within the aorta of the horse, considered the two to be quite different in their origin. The abrupt wave in the aortic trace he attributed to the closure of the aortic valves, but he believed that the dicrotic wave, as seen in the peripheral arteries, was produced in those arteries themselves. If, however, in a patient who has aneurismal dilatation of the aorta, a series of tracings be obtained from the aorta, subclavian, brachial, and radial arteries, the change in shape of the dicrotic wave is found to be quite a gradual and progressive one, and a still further transformation of the same kind is found to have taken place in the dorsalis pedis.

The latest view held by Marey as to the causation of the dicrotic wave, as explained in his recently published work (' Physiologie Expérimentale,' 1875), is that it is a

wave which proceeds from the centre towards the periphery, and that it is caused by an oscillation depending chiefly upon the inertia of the fluid and the velocity with which it is thrown into the arteries, but that this oscillation takes place not in the aorta, but at the commencement of each individual artery, as it arises from the aorta. Amongst other objections to this theory I would state that I have found the dicrotic wave, for instance, in the abdominal aorta to be as well marked, or nearly as well marked, as in the femoral artery of the same person. My own view, as far as regards the chief origin of the dicrotic wave, differs from Marey's only in this, that I maintain that the oscillation takes place, in great measure at any rate, at the commencement of the aorta itself, and that it is modified in an important degree by the action of the aortic valves. I think that this view receives a strong confirmation in the almost entire absence of the dicrotic wave, in traces taken near to the heart as well as in those from more distant points, in some cases of very free aortic regurgitation. Of this the most striking instances among the tracings appended to this paper are figures 9, 10, and 11 in Plate XVI, representing the carotid, radial, and dorsalis pedis pulses, figures 5 and 6 in Plate XVI, representing the subclavian and radial pulses, and figure 12 in Plate XVI, representing the radial pulse, the last being taken from a case in which acute fever was present, the temperature being 104°.

There is another circumstance about the dicrotic wave which it is important to note, namely, the change of its relative position. In all cases it is found that, while the interval between the commencement of the primary and that of the dicrotic wave does not greatly vary, there is a marked and progressive increase, as the pulse-wave recedes from the heart, in the interval measured either from the commencement or summit of the primary wave to the summit of the dicrotic wave (c). This is seen in the series of normal tracings from carotid, radial, and dorsalis pedis arteries shown in Pl. XV, figs. 1, 2, and 3,

and also in the corresponding tracings from a case of
aortic regurgitation (Pl. XVI, figs. 1, 2, and 3). The
results which I have obtained are in this respect also con-
firmed by a comparison with the tracings obtained from
different arteries by Wolff and by Professor A. H. Garrod.
Since, then, the velocity of a wave must be regarded as
being the velocity of its summit, it is proved that the
dicrotic wave is transmitted more slowly than the primary.
This is readily explained by the same cause which has
been already found to account for the change in shape of
the primary wave. For the tension is much less at the
summit of the dicrotic than at the summit of the primary
wave, and the velocity of the former wave is therefore
less. The difference of tension will of course be greater
when the pressure is unusually low during diastole, and
therefore we may expect that in such cases the dicrotic
wave will fall behind more rapidly. Accordingly we find
that in the radial pulse shown in Pl. XV, fig. 14, taken from
a patient whose arteries were extremely lax, the interval
between the commencement of each beat and the summit
of the dicrotic wave is increased by more than one half, as
compared with the same interval in the carotid pulse
(fig. 13). So great is this increase that, supposing it to
take place at the same rate in the pulse-wave which
proceeds to the foot, we should expect to find that, in the
dorsalis pedis, the dicrotic wave had been entirely lost
upon the succeeding beat. This seems indeed to have
been actually the case, for in the curve of the dorsalis
pedis (fig. 15) no dicrotic wave is visible, and its former
place is occupied by a flat interval. The difference of
tension and therefore difference of relative velocity will
be still greater in many cases of aortic regurgitation, and
hence it is possible that the absence of the dicrotic wave
in traces of such pulses, taken at a distance from the
heart, as in the dorsalis pedis artery, may sometimes be
due, not so much to its having never existed as to its
having become lost upon the succeeding systolic wave.

There are some radial pulses, and still oftener pulses

taken from arteries nearer the heart, in which the tricrotic wave (see Pl. XV, fig. 2 D) is seen following the dicrotic wave. It appears in general to be chiefly formed by the second member of that series of oscillations of which the dicrotic wave is the first, and it has the same, or a still greater, relative slowness of transmission. Accordingly it is never seen in the dorsalis pedis pulse (see Pl. XV, fig. 3), but it has always become lost upon the succeeding beat, before the pulse-wave has arrived at that distance from the heart.

I have introduced one radial pulse-tracing, namely, that in Pl. XVI, fig. 8, as a contrast to the tracings of aortic regurgitation. It is one which indicates considerable aortic obstruction, without any perceptible regurgitation. Cases in which such a result is obtained by the sphygmograph are infinitely more rare than those in which only a systolic and no diastolic murmur is heard by the stethoscope. It will be seen that there is a very large tidal wave, which rises far higher than the summit of the primary wave, and indicates the prolonged ventricular contraction; the pulse is irregular and very slow ; and there is a marked dicrotic wave, to be associated with the fact that there was no free reflux through the aortic orifice.

I think that an interesting inference may be drawn from the characters of the water-hammer pulse, as revealed by the sphygmograph, with regard to the causation of that wasting, pallor, and cachexia which so distinguish aortic regurgitation from other forms of heart disease, and which often make the subject of it somewhat resemble a patient suffering from phthisis. One of the most important of the natural functions of the elastic coats of arteries is to render equable the stream of blood, which when it starts from the heart is interrupted, and it is probable that this is accomplished not so much in the very smallest arteries—whose coats are chiefly muscular, and have the function of regulating the blood supply at different times—as in those of larger size. The uniformity

thus produced in the current through the capillaries is no doubt of essential importance for the due progress of that exosmosis and endosmosis between the vessels and the tissues upon which nutrition depends. Now, it has been shown that, when there is any considerable aortic regurgitation, so far from the larger arteries rendering the blood stream more uniform, it becomes for a considerable distance progressively more unequal, and the point of maximum inequality is often not far distant from the smallest arteries. In such cases it can hardly fail to be the result that there is perceptible inequality even in the current through the capillaries, and it is not therefore wonderful that there is more effect upon nutrition, as compared with other results of disease, in aortic regurgitation than in other valvular imperfections.

It may be well to add, to avoid any risk of misconception, that throughout this paper I have spoken of as the systolic portion of the pulse-curve that part which corresponds to the systole of the heart, not that during which the artery is contracting. Similarly, by the term diastolic portion I mean that part of the curve which corresponds to the diastole of the heart. The junction of the two portions is nearly, but not precisely, marked by the aortic notch, or commencement of the dicrotic wave, its precise position being somewhat anterior to that point.

DESCRIPTION OF PLATES XV and XVI.

The tracings have been copied by photo-lithography, and are represented of the original size. The traces from large arteries, the subclavian, carotid, or femoral, were taken with an amplifying power less than is usual in the sphygmograph, and the extent of amplification in vertical height of each of such traces is indicated in the description of the figures. For the radial and dorsalis pedis arteries the ordinary sphygmograph was used, having an amplifying power of about 90. In many of the curves measuring lines are drawn to show the relative intervals of time occupied by the main upstroke in different cases. These are arcs of circles, having a radius equal to the length of the recording lever of the sphygmograph, and are so drawn that the centre of each arc corresponds exactly in relative position to the axis of motion of the recording lever, at the time when the tracing was taken. In this way all points on the curve may be reduced to a horizontal line for purposes of exact measurement. All the traces of aortic regurgitation were taken from patients in whom the valvular disease appeared to have been due to rheumatism, and not to extension to the valves of disease affecting the aorta, in order to exclude, as far as possible, from the curves any effects due to atheroma of the arteries. The letter B in the figures denotes the tidal (or predicrotic), C the dicrotic, and D the tricrotic wave. In the comparative tracings of radial and dorsalis pedis arteries both curves were taken while the patient was recumbent, except where it is otherwise specified.

PLATE XV.

Fig. 1.—From the carotid artery of a healthy man, aet. 26. Amplified 30 times.

Fig. 2.—From the radial artery of the same person. Pressure 4 oz. The curve is not exactly typical of a normal radial pulse, for it is of unusual amplitude, approximating in this respect to the pulses of aortic disease, and it shows the secondary waves with unusual distinctness. It represents rather the pulse of an athlete. The trace was obtained while the person was in a sitting posture, and the pulse is therefore quicker than those in figs. 1 and 3, which were taken while he was recumbent.

Plate XV.

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Plate XV.

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Fig. 3.—From the dorsalis pedis artery of the same person. Pressure 3 oz.

Fig. 4.—Thomas G—, æt. 20. Aortic regurgitation. Radial pulse-curve. Pressure 3 oz.

Fig. 5.—From the same pulse at a pressure of 4½ oz.

Fig. 6.—James B—, æt. 29. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz.

Fig 7.—From the femoral artery of the same patient. Amplified 40 times.

Fig. 8.—From the dorsalis pedis artery of the same patient. Pressure 2 oz.

Fig. 9.—Tracing taken from a schema of bifurcating elastic tubes representing the arterial system, and adapted to an artificial heart of india rubber, which was worked by means of the hand. This curve was taken from the main trunk of the schema, not far from the valves. Amplified 20 times.

Fig. 10.—Tracing simultaneous with fig. 9, taken from a small tube of the schema at a distance of 4½ feet from the former. Amplified 40 times. The clockwork movement was very slightly slower than in fig. 9.

Fig. 11.—James J—, æt. 60. Aortic regurgitation. Radial pulse-curve. Pressure 4 oz. Only a systolic murmur at the base was audible at the time, but the pulse had a marked aortic quality to the finger.

Fig. 12.—John S—, æt. 17. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz. The pulse to the finger had an evident, although not a very marked, water-hammer quality.

Fig. 13.—Th. J—, æt. 31. Tracing from the carotid artery. Amplified 30 times. The patient was suffering from phthisis, and had a temperature of 101·8°.

Fig. 14.—From the radial artery of the same patient. Pressure 3 oz.

Fig. 15.—From the dorsalis pedis artery of the same patient. Pressure 3 oz.

Fig. 16.—James T—, æt. 20. Aortic regurgitation. Radial pulse-curve. Pressure 2 oz.

Fig. 17.—John P—, æt. 17. Aortic regurgitation. Radial pulse-curve. Pressure ½ oz. The patient was suffering from pneumonia, and had a temperature of 103·1°.

PLATE XVI.

Fig. 1.—Mary T—, æt. 22. Aortic regurgitation. Tracing from the carotid artery. Amplified 40 times. The pulse was manifestly double to the finger, the second beat preceding the second sound of the heart, and therefore not being due to the dicrotic wave.

Fig. 2.—From the radial artery of the same patient, the arm being horizontal. Pressure 3½ oz.

Fig. 3.—From the radial artery of the same patient, the arm being raised. The pressure was 2 oz., higher pressures not giving so ample a trace. The water-hammer quality was very marked to the finger when the arm was raised, although the pulse was not a full one.

Fig. 4.—From the dorsalis pedis artery of the same patient. Pressure 2 oz.

Fig. 5.—Thomas L—, æt. 30. Aortic regurgitation. Tracing from the subclavian artery. Amplified 20 times.

Fig. 6.—From the radial artery of the same patient. Pressure 5 oz.

Fig. 7.—From the dorsalis pedis artery of the same patient. Pressure 4 oz.

Fig. 8.—Henry G—, æt. 54. Aortic obstruction. Pressure 4 oz.

Fig. 9.—George G—, æt. 33. Tracing from the carotid artery. Amplified 20 times.

Fig. 10.—From the radial artery of the same patient. Pressure 4 oz.

Fig. 11.—From the dorsalis pedis artery of the same patient. Pressure 4 oz. In this patient, when recumbent, the water-hammer quality was to the finger far more marked in the dorsalis pedis than in the radial artery. There was also an unusually marked interval of time between the pulsation in the different arteries.

Fig. 12.—Charles P—, æt. 18. Aortic regurgitation. Radial pulse-curve. Pressure 4½ oz. The patient was suffering from incipient pneumonia (which quickly proved fatal), and his temperature was 104°. This tracing was taken with the arm horizontal.

Fig. 13.—From the radial artery of the same patient, the arm being raised. The pressure was 3 oz., higher pressures not giving so ample a trace. The suddenness of the pulse was very marked to the finger when the arm was elevated.

Fig. 14.—William T—, æt. 24. Aortic regurgitation. Radial pulse-curve taken with the arm horizontal. Pressure 3 oz.

Fig. 15.—From the radial artery of the same patient, the arm being raised. Pressure 3 oz.

Plate XVI.

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OBSERVATIONS

ON

BOX (BUXUS SEMPERVIRENS),

WITH ESPECIAL REFERENCE TO THE TRUE NATURE OF

TETANUS.

BY

SYDNEY RINGER, M.D.,
PROFESSOR OF THERAPEUTICS AT UNIVERSITY COLLEGE;

AND

WILLIAM MURRELL, L.R.C.P.,
DEMONSTRATOR OF PHYSIOLOGY AT UNIVERSITY COLLEGE.

Introductory and Experimental.

WE were induced to commence an investigation of the physiological action of box from the reputation it has acquired in the treatment of hydrophobia, and the frequency with which it enters into the composition of nostrums having obtained a reputation for the cure of this disease.

The curious, and at first sight contradictory, phenomena observed in frogs which had been injected with box led us to deviate from our original plan, and to make the experiments here detailed in the hope of thrôwing some light on the true nature of tetanus.

As yet we have had but little experience of the use of

box clinically, and it is not our intention in the present communication to offer any observations as to its employment in the treatment of disease, or its value as a therapeutical agent.

All the experiments recorded in this paper were made on frogs. We used two extracts of box; one dilute, containing in each minim the active principle of one grain of the dried leaf, and a concentrated extract three times this strength. The drug was introduced under the skin in the neighbourhood of the posterior lymph hearts by means of a hypodermic syringe. In all cases where the animal survived for some hours care was taken to keep it moist, and when not actually under observation it was placed on a glass plate in a flat covered dish containing a little water.

The extract was prepared for us by Mr. Gerrard, teacher of pharmacy at University College.

We will first describe a typical case. Two minutes after the injection the frog became dull and its movements slow, and this condition increasing, in five minutes it could not turn over when laid on its back. In twenty minutes slight tetanus occurred, at first provoked only by strong stimulation, a weaker one exciting only coördinated reflex action. At first, too, though for a very short time, the tetanus was limited to the irritated limb. Soon, however, normal reflex action was entirely replaced by tetanic convulsions, and the tetanus rapidly increased, becoming severe thirty-eight minutes from the commencement of the experiment, and remained at its height for six minutes, and then rapidly declined. (So rapid indeed is the declension sometimes, that on one occasion where the paroxysm lasted three quarters of a minute, afterwards, when re-excited, the paroxysm lasted only a quarter of a minute, and after another five minutes interval only two seconds.) The tetanus continued very weak for an hour, during which time stimulation excited only a feeble tetanic paroxysm, speedily subsiding, and tetanus could not be again produced till the animal had rested awhile. As the

tetanus declined stronger irritation and a longer rest were necessary to the production of a paroxysm, whilst the attacks grew gradually feebler and feebler, till they diminished to a mere quiver of the muscles. It was evident that tetanus and paralysis were progressing together, the paralysis gradually increasing, overpowering the tetanus, and finally abolishing it.

In this case first loss of power occurred, which appeared to us more like loss of voluntary than reflex power; next, rather severe tetanus accompanied by paralysis. We had, in fact—

1. Tetanus with increased action—or strong tetanus.
2. Tetanus with depressed action—or weak tetanus.
3. Complete paralysis.

The foregoing account represents a typical experimental case, yet we met with many exceptions, the symptoms running a somewhat different course. Thus in certain cases, due as we shall show to the degree of concentration of the extract, the tetanus was very slight, so slight, indeed, that in each tetanic convulsion we think the discharge of nervous force in the cord must have been less than in a vigorous normal (coördinated) reflex act. In other instances the tetanus was still slighter, so that the nervous discharge must have been less than that in a coördinated reflex act. Here then we had simply : 1. Tetanus with depressed action. 2. Complete paralysis.

In all the cases, even when the tetanus was severe, a stage was reached when becoming no stronger than a normal reflex act, the tetanus ultimately became even weaker than a normal reflex act.

In other cases no tetanus occurred, but simply progressive paralysis, which at last became complete. These varying results were produced by the same dose of the poison in frogs of the same weight, an apparent discrepancy which our further investigations clear up. Thus a very concentrated solution of the extract excites strong tetanus, whilst the same dose diluted excites either no tetanus, or very little, as the accompanying tables exemplify. The

frogs included in the first table were injected under the skin of the back in various doses with a solution the strength of three grains of the dried leaf in one minim of water, and with two exceptions this preparation produced strong tetanus. In the *first* column we give the date; in the *second* the weight of the frog; in the *third* the dose of the extract, giving the quantities in grains of the leaf; in the *fourth* the degree of tetanus induced; in the *fifth* the time of commencement of the tetanus; in the *sixth,* the time from its commencement when the tetanus begun to decline; and in the seventh the duration of the tetanus. The table is divided into two parts, in the first part the frogs were merely poisoned, in those in the second part the cord was divided opposite the occipito-atlantal membrane, and then a peg was passed upwards into the skull, thus destroying the medulla and brain (brainless frogs).

Table I.—*Unmutilated frogs.*

Part 1.

Date.	Weight in grammes of frog.	Dose of leaf in form of extract.	Degree of tetanus.	Tetanus first occurred in	Tetanus began to decline from its commencement.	Duration of tetanus.
April 7	...	30 grains	Strong	13 minutes		
„ 7	...	12 „	„	19 „		
„ 11	38	9 „	„	22 „	35 minutes	2 h. 25 m.
„ 12	...	12 „	„	27 „	16 „	
„ 12	...	27 „	„	18 „	15 „	1 h. 8 m.
„ 13	30	30 „	„	10 „	19 „	1 h. 8 m.
„ 20	25	9 „	„	16 „	18 „	2 h. 35 m.
„ 20	32	15 „	„	18 „	16 „	1 h. 20 m.
„ 20	24	6 „	„	29 „	11 „	2 h.
„ 20	26	9 „	„	19 „	16 „	1 h. 43 m.
„ 20	19	9 „	„	16 „	17 „	1 h. 53 m.
„ 20	23	9 „	„	18 „	20 „	1 h. 36 m.
Average	27*	12* grains		19 minutes	18 minutes	1 h. 45 m.

* These averages are calculated only where both weight and dose are given.

TABLE I (*continued*).—*Brainless frogs.*

Part 2.

Date.	Weight of frog in grms.	Dose of leaf in form of extract.	Degree of tetanus.	Tetanus first observed in	Tetanus began to decline from its commencement	Duration of tetanus.	Loss of reflex power preceding tetanus.
April 7	28	27 grains	Strong	16 min.	...	1 h. 37 m.	
,, 7	28	27 ,,	,,	30 ,,	...	1 h. 10 m.	
,, 11	25	24 ,,	,,	32 ,,	...	2 h. 39 m.	
,, 12	...	27 ,,	,,	24 ,,	...	...	5 min.
,, 12	25	24 ,,	,,	32 ,,	...	1 h.	
,, 21	27	9 ,,	,,	47 ,,	53 min.	1 h.	
,, 21	29	12 ,,	,,	33 ,,	27 ,,	1 h. 30 m.	5 ,,
,, 21	28	12 ,,	,,	36 ,,	56 ,,	1 h.	
,, 21	27	12 ,,	,,	40 ,,			
,, 22	22	9 ,,	,,	26 ,,	29 ,,	1 h. 50 m.	10 ,,
,, 22	28	9 ,,	,,	31 ,,	41 ,,	50 m.	5 ,,
,, 24	20	9 ,,	,,	21 ,,	37 ,,	1 h. 25 m.	5 ,,
,, 24	31	9 ,,	,,	34 ,,	24 ,,	1 h. 21 m.	8 ,,
,, 24	30	9 ,,	,,	25 ,,	32 ,,	...	21 ,,
,, 24	21	9 ,,	,,	24 ,,	28 ,,	1 h. 10 m.	5 ,,
May 1	22	9 ,,	,,	21 ,,	28 ,,	1 h. 43 m.	10 ,,
,, 1	...	9 ,,	Very weak	14 ,,	...	...	5 ,,
,, 1	19	9 ,,	Very strong	20 ,,	43 ,,	3 h.	10 ,,
,, 1	19	9 ,,	Slight	25 ,,	...	1 h. 20 m.	10 ,,
,, 2	27	9 ,,	Strong	19 ,,	44 ,,	1 h. 40 m.	8 ,,
Average	25	4 grains		27 min.	37 min.	1 h. 31 m.	8 min.*

* Excluding the 21 minutes, average 7 minutes.

TABLE II.—*Exemplifying the effect of the dilute extract, each minim containing the extract from one grain of the dried leaf.*

Date.	Weight of frog in grammes.	Dose equivalent to dried leaf.	Amount of tetanus.	Tetanus first appeared.	Duration of tetanus.
April 13	31	5 grains	Moderate	1 h. 15 m.	50 m.
„ 13	35·5	10 „	None		
„ 13	38	15 „	„		
„ 13	39	20 „	Slight	52 m.	1 h. 11 m.
„ 19	30	5 „	None		
„ 19	32	10 „	Slight	1 h. 33 m.	20 m.
„ 19	29	15 „	Moderate	56 m.	1 h. 35 m.
„ 19	29	20 „	None		
„ 19	30	10 „	Moderate	39 m.	1 h. 50 m.
„ 19	34	10 „	None		
„ 19	30	13 „	„		
„ 20	26	9 „	Very slight	1 h. 26 m.	10 m.
„ 20	21	15 „	None		
„ 20	24	6 „	„		
„ 20	30	9 „	Very slight	57 m.	1 h. 5 m.
Average	29	11 grains		1 h. 16 m.	1 h.

In the fifteen cases in the last table, in eight no tetanus occurred, moderate tetanus in three, and very slight tetanus in four. The dilute solution produced generally very slight tetanus, each paroxysm being, so far as we could estimate, weaker than a normal (coördinated) reflex act, and in several cases much weaker.

In the second table in every instance almost complete loss of voluntary and reflex power preceded the onset of the tetanus; indeed in two cases the paralysis was complete before the tetanus set in.

We suggest that the difference in the effect produced by the two solutions is owing to the quicker absorption of the concentrated solution. Its specific gravity being greater, it will pass by the law of diffusion more quickly into the blood.

The preceding tables show that the tetanus when well marked begun on an average in 19 minutes, continued

TABLE III.—*Showing time of onset of loss of power in frogs produced by box.*

Number of frog.	Weight of frog in grammes.	Dose of leaf in form of extract.	Strength of preparation.	Loss of power begun in
1	30	5 grains	Dilute	5 minutes
2	32	10 ,,	,,	5 ,,
3	29	15 ,,	,,	3 ,,
4	29	20 ,,	,,	3 ,,
5	30	10 ,,	,,	2 ,,
6	34	10 ,,	,,	2 ,,
7	30	13 ,,	,,	2 ,,
8	25	9 ,,	Concentrated	3 ,,
9	32	15 ,,	,,	1 ,,
10	26	9 ,,	,,	3 ,,
11	26	9 ,,	Dilute	1½ ,,
12	21	15 ,,	,,	2 ,,
13	24	6 ,,	,,	2 ,,
14	30	9 ,,	,,	2 ,,
15	19	9 ,,	Concentrated	1 ,,
16	23	9 ,,	,,	3½ ,,
Average	27·5	10·8 grains		2½ minutes

To ascertain if all or part of this loss of power is due to the action of the box on the brain or the medulla, we divided the spinal cord, opposite the occipito-atlantal membrane in fifteen frogs, and then passed a wooden peg into the skull, destroying the medulla and brain (brainless frogs). When the effects of shock had passed off, we injected under the skin of the back the concentrated solution of the extract each minim containing the extract from three grains of leaf. Tetanus first appeared in from 14 to 47 minutes, giving an average in the twenty cases of 27 minutes (see tables on pp. 393 and 394). Shortly before the onset of the tetanus, we noticed slight but distinct diminution of reflex power, so that we could fortel the occurrence of the tetanus; the loss of reflex power generally preceding the tetanus, about five minutes, though in one case there was an interval of twenty-one minutes ; the average being eight minutes, or excluding the case of twenty-one minutes, the average was seven minutes. We have shown that in

unmutilated frogs, loss of power occurs in two and a half minutes; tetanus occurs on an average in nineteen minutes. Where the influence on the cord and medulla is prevented, in pegged (brainless) frogs the tetanus is preceded by loss of reflex power for seven minutes ; therefore the loss of power occurring during the twelve first minutes must be due to the influence of the poison on either the brain or the medulla.

To determine whether the loss of reflex action shortly preceding the tetanus in pithed frogs is a natural decline or is due to the action of the drug, we performed some comparative experiments by poisoning each alternate frog, and watching the reflex action as the poisoned and unpoisoned frogs lay side by side. We find that in unpoisoned frogs, if the animals are kept moist, reflex action continues undiminished for thirty hours, indeed sometimes for fifty hours, and persists from fifty to a hundred hours. For further details concerning the duration of reflex action we refer our readers to the section on tetanus p. 414.

We may draw attention here to one fact connected with the duration of reflex action in brainless frogs, which explains we think the variable effects of box and gelseminum on the cord. Thus, in some instances reflex action had ceased in 23 hours, whilst in others it lasted for 90 to 100 hours. There must be therefore naturally considerable differences in the condition of the cord, and hence the cord in one frog will be quickly and easily paralysed, whilst another may require a larger dose and a longer time.

In brainless frogs we found that box produced tetanus, preceded for a short time, as we have said, by diminution of reflex power, which further diminishing at last becomes complete. This loss of reflex power and tetanus must depend on the effect of the drug, either on the cord, nerves, or muscles. It will be convenient to treat separately of tetanus and paralysis.

Tetanus is certainly due to the action of the drug on the spinal cord for—1. There is no example of the induc-

tion of tetanus through the nerves or muscles. 2. If before poisoning, the abdominal aorta or one iliac is tied, thus protecting from the action of the poison the part below the ligature, yet these parts become tetanised as strongly as those subjected to the direct action of the poison. We proved this by eleven experiments; four times we tied the abdominal aorta, and seven times one iliac artery, verifying by post-mortem examination the efficiency of the ligatures. 3. If the sciatic nerve is divided, the other structures being left intact, the muscles of the extremity with the divided nerve are not tetanised. We verified this statement by three experiments, two on frogs, one on a toad. In each animal we divided the sciatic nerve of the left leg, and then injected under the skin of the back four minims of the stronger extract of box. In about fifteen to seventeen minutes, tetanus set in, which did not involve the muscles of the leg and foot of the left extremity, though some of the thigh muscles were affected. Irritation of the left leg failed to excite a paroxysm of tetanus.

Box likewise paralyses the reflex function of the cord. To prove this we experimented on four frogs, by tying the abdominal aorta and then poisoning the animal by injecting under the skin of the back three minims of the concentrated solution (equal to nine grains of leaf). The lower extremities were thus, of course, effectually protected from the action of the poison, and yet paralysis progressed just as in frogs, unprotected by a ligatured aorta. The following table shows the result of these experiments.

Date.	Weight of frog in grammes.	Dose.	Paralysis.	Tetanus.	First appeared.	Tetanus lasted
April 10	30	6 grains	Complete	Strong	33 min.	48 min.
May 1	25	9 ,,	,,	,,	16 ,,	87 ,,
,, 1	20	9 ,,	,,	Very slight		
,, 1	19	9 ,,	,,	None		
Average	23	8 grains			24 min.	67 min.

That both tetanus and paralysis are produced by the action of the drug on the cord is proved in our experiments regarding the influence of box on the motor nerves. In these experiments, seven in number, we tied one iliac artery, and then poisoned the animal, and in every instance save one, paralysis as complete, and tetanus as strong, seized the ligatured and protected as well as the unligatured and unprotected leg. As the muscles and motor nerves were protected by ligature, it is evident that the paralysis was not due to the action of the drug on these structures.

It is well known that some drugs as calabar bean, which paralyse through their action on the spinal cord, do likewise to a slight extent paralyse the motor nerves. Box we conclude does not affect either the motor nerves or the muscles; for did it in any degree paralyse these structures, then on tying one iliac artery before poisoning, and thus protecting the nerves and muscles of one limb from the poisoned blood, tetanus should be more marked in the protected, and paralysis should progress more rapidly in the unprotected limb. For the poison depressing the motor nerve of the unligatured limb would diminish its conducting power, and hence lessen the tetanic contractions of its muscles. This is well exemplified by slightly pressing or stretching the nerves of one leg, when the muscles of that leg are less powerfully contracted during the tetanic spasm. Moreover, if box affects either the motor nerves or muscles the paralysis in

the unprotected would progress more rapidly than in the protected leg. Again, if box affects either the nerves or muscles before the spinal cord, then loss of reflex action should occur earlier in the unprotected than in the protected limb. Now, we find as the result of seven observations given in the subjoined table, in which one iliac artery was tied, that with only one readily explicable exception, tetanus was equally well marked, that paralysis progressed equally, and was complete at the same time in both legs; moreover, the loss of reflex action begun simultaneously in both legs.

Table showing comparative amount of tetanus and paralysis in the legs produced by box after ligature of the iliac arteries.

Experiment	Date.	Weight of frog.	Dose of extract.	Proportion of dose to weight of frog.	Vessels tied.	Result.
XXXIV	1876. April 25	Grms. 76	Grains. 30	...	Lt. iliac	Tetanus less in left leg than in right
XXXV	„ 25	65	27	...	Rt. iliac	Tetanus slightly less in left leg than in right
XXXVI	„ 26	25	9	...	„	Tetanus equal
LV	May 2	24	12	...	„	Tetanus equal; loss of reflex action equal
LVI	„ 2	25	12	...	„	Tetanus equal; reflex action slightly greater in right leg
LVII	„ 2	22	12	...	„	Tetanus equal; reflex action slightly greater in right leg before injection; subsequently no difference could be detected
LVIII	„ 2	19	9	...	„	Tetanus equal; reflex action equal in both legs
Average		38	14	...		

In the last column we compare the amount of tetanus and paralysis in the protected and unprotected limbs.

This table shows that in seven. frogs operated on successfully, one only exhibited any excess of tetanus, though slight, in the protected limb.

It appears then, as we have already stated, that box exerts no influence on the motor nerves or muscles. In the exceptional case just mentioned, it happened in the course of the operation, that a slight injury was inflicted on one of the abdominal nerves, thus impairing its conductivity and readily accounting for the predominance of tetanus in the limb with its uninjured nerve.

In all our operations on the abdominal vessels, we took every care to avoid stretching or even touching the adjacent nerves, but occasionally they lay in such close contact with the artery that it was extremely difficult to avoid injuring them.

With the view, however, of setting this question beyond the possibility of doubt we performed eight additional experiments, in which we resorted to a somewhat different mode of procedure. We will give the details of one of the most conclusive of these observations. We tied the right iliac artery of a large male German frog weighing sixty-five grammes. Fortunately very little blood was lost, and the animal seemed but little affected by the operation, jumping about actively on being released. An injection of extract of box equivalent to twenty-seven grains of the dried leaf was then administered in the neighbourhood of the posterior lymph hearts, care being taken that none of the fluid escaped through the incision made for the operation. Tetanus and paralysis were produced in due course, but into the details of these phenomena we need not now enter. Fifty minutes after the administration of the drug, when the paralysis was complete, and the tetanus had entirely ceased, the thighs were opened and the sciatic nerves exposed. A piece of very thin flat glass was then passed under each nerve so as to completely isolate ; they were stimulated with a

pair of electrodes in connection with a Du Bois Reymond's induction coil, and a one-celled Daniell's battery, every care being taken to apply the excitor to each nerve in exactly the same manner and under identically the same conditions. We occasionally used a powerful shock, but as a rule we worked with the current of minimum intensity just adequate to produce the slightest perceptible quiver in the muscles to which the nerves were distributed.

It would be wearying and unprofitable to give the actual details of each single observation. We compared the condition of the nerves as regards excitability twenty-five times during a period of a little over four hours, and were unable in any instance to detect the slightest difference in their excitability.

This case alone would, we venture to think, prove conclusively that box exerts absolutely no influence on the motor nerves. There are in addition six other cases in which we successfully tied one iliac artery, and in these the most careful observation failed to detect the existence of even the slightest or most transitory difference in the condition of the nerves.

An examination of the accompanying table will show at a glance the results obtained in this series of experiments. In the first and second cases we made a considerable number of comparative observations, so as thoroughly to satisfy ourselves as to the correctness of our conclusions, but the actual number was not noted.

Table showing condition of sciatic nerves in frogs poisoned by box after ligature of one of the iliacs.

No. of experiment.	Date.	Weight of frog.	Vessels tied.	Grms. injected	Relation of dose to weight of animal.	Time of observation after injection.	No. of comparative observations.	Result.
	1876.	Grms.						
I	Ap. 12	34	R. iliac	9	...	1 h. 41 m.	P	No difference
II	„ 12	30	„	5	...	1½ h.	P	„
III	„ 25	65	„	27	...	1 h.	2	„
						1 h. 5 m.	2	„
						1 h. 10 m.	8	„
						1 h. 15 m.	1	„
						1 h. 20 m.	8	„
						1 h. 30 m.	1	„
						1 h. 45 m.	8	„
						2 h.	1	„
						2 h. 30 m.	8	„
						2 h. 45 m.	1	„
						3 h. 10 m.	2	„
						3 h. 30 m.	1	„
						4 h.	1	„
						4 h. 10 m.	1	„
IV	„ 26	25	„	9	...	9 h. 40 m.	13	„
V	May 2	24	„	12	...	4 h.	Many	The right sciatic nerve conducts better than the left, although the difference is slight
						5 h.	7	The right nerve still acts better than the left
VI	„ 2	25	„	12	...	6 h.	6	No difference
VII	„ 2	22	„	12	...	2 h.	10	„
VIII	„ 2	19	„	9	...	1 h.	8	„

In one instance there was a decided difference in the excitability of the two sciatics, the nerve of the protected limb acting distinctly more energetically than that of the other. What value are we to attach to this exceptional result? In answer to this question we cannot do better than detail another experiment in which the operation for ligature of the iliac artery was performed. The box was given as usual, and immediately after the decline of the tetanus and the occurrence of complete paralysis the

two sciatic nerves were exposed and an investigation was
made of their power of conducting electrical stimuli as
measured by the contractions of the muscles of the limb.
The excitability exhibited by the nerve of the left leg was
clearly greater than that of the right. The observation
was made several times with varying strengths of current,
and the result was in every case the same. At the post-
mortem examination it was found that neither iliac artery
had been tied !

This experiment demonstrates the fact that occasionally
a difference normally exists in the relative activity of the
two sciatic nerves. It may, we think, be fairly concluded
that our exceptional case belongs to this category, and
that the difference in the condition of the two nerves
would have been equally apparent had we resorted to no
operative procedure and had left the arteries intact.

Does box in any degree affect the muscles ? In the
experiments just detailed we tested the muscles of the
opposite legs, the muscles of one extremity being protected
from the action of the poisoned blood by ligature of its
artery. The apparatus used to stimulate the muscles
was that employed in the investigation of the motor nerves.
The electrodes, however, were placed in actual contact
with the muscular tissue. The general mode of procedure
was in both investigations identical. In no instance
were we able to discriminate the slightest difference either
in the strength or the rapidity of contraction of the corre-
sponding muscles of the two limbs. We conclude there-
fore that box exerts no direct influence on the muscular
tissue.

We next proceeded to ascertain whether box poisons
the afferent nerves, for if this drug had induced depres-
sion of these nerves, before the cord became tetanised,
this condition would throw light on the paralysis pre-
ceding tetanus, for the depressed afferent nerves would
convey to the cord a weaker impression, and the reflex acts
would consequently be weaker. We find that the afferent
nerves are not paralysed by box, and we base this conclu-

sion on the following experiment. After tying the iliac artery of one leg and then poisoning the animal by injecting the drug under the skin of the back, we tested reflex action in both posterior extremities by holding the animal by its toes, and we found always that reflex function remained equally good in both legs. Now, if the drug poisons the afferent nerves the impressions conveyed along the poisoned limb should then be less active than those passing through the unpoisoned limb, and reflex action in place of remaining unaffected should be less perfect when the animal is suspended by the poisoned extremity. The foregoing experiment, however, being insufficiently delicate to settle this important question, we sought more convincing evidence, and four times we repeated the following experiments. We divided the cord just below the medulla, and then destroyed the medulla and brain by passing a wooden peg into the cavity of the skull. We then tied one iliac artery carefully, closing the wound with ligatures. We next determined the weakest induction currents capable of exciting reflex action in either hind leg, and then injected under the skin of the back four mimims of the concentrated extract of box. Every three or five minutes we tested the weakest currents adequate to excite reflex action in the posterior legs. As the reflex function declined, the same strength of currents would still excite in each limb an equal amount of reflex action in the poisoned as in the unpoisoned limb, till the abolition of all reflex action. We conclude, therefore, that box exerts no influence on the afferent nerves.

Having thus shown that box tetanises and paralyses by its effects on the cord, and that the afferent and motor nerves and muscles are unaffected, we wish to draw particular attention to a fact several times referred to in this investigation—that in pegged frogs, when the drug cannot of course act on the brain, after the injection of the concentrated solution of the extract, tetanus is preceded for about six minutes by loss of reflex power in the cord, and that this loss is manifested even where the subse-

quent tetanic convulsions are severe, and the nervous discharge in the cord is much greater than occurs in a normal (coördinated) reflex act; hence, according to the prevailing notion, the cord is said to be excited by the medicine. Very soon after the onset of the tetanus and even before it becomes severe the coördinated reflex power of the cord rapidly declines. Thus we have first marked loss of reflex power and then strong tetanus. This apparent contradiction—this loss of coördinated reflex power preceding tetanic convulsions and persisting in the intervals of the paroxysms—might be due, we thought, to the depressing influence of the drug on the motor or afferent nerves or muscles before the excitation of tetanus in the cord; but having shown that box does not affect these structures, it is clear that the drug must first lessen reflex action in the cord and then produce tetanus, the coördinated reflex power itself meanwhile diminishing. This apparent contradiction we have attempted to reconcile in the section on tetanus.

In twelve observations with unmutilated frogs, as we have already shown, energetic tetanus lasts only a short while, beginning to decline on an average in eighteen minutes, and in ten brainless frogs on an average in thirty-nine minutes, then it grows rapidly weaker and so continuing for an hour to an hour and a half; it then ceases and all reflex action is abolished. Is this due to the natural cessation of the functions of the cord, or to exhaustion from the tetanus, or to the medicine? It is not due to the natural death of the cord, for after dividing the cord opposite the occipito-atlantal membrane we found that reflex action continues unimpaired for thirty or forty hours. (For further remarks on the persistence of reflex action after death, see the section on tetanus.)

Nor is it due to exhaustion from the tetanic convulsions, since strychina induces far intenser tetanus, lasting several days, whilst the tetanus from box is much less severe and lasts only an hour or two and then ends in complete paralysis. The paralysis of the cord is therefore due to the direct action of the box.

Box, then, paralyses and tetanises the spinal cord. Are these results due to the action of the drug on Setschenow's reflex inhibitory centre; in other words, is the paralysis due to stimulation or the tetanus to paralysis of this centre? Clearly not, for both paralysis and tetanus occurred after section of the spinal cord below this centre.

The following is a summary of our conclusions:

1. Box produces loss of power by its effects either on centres of volition or on the motor centres of the brain.

2. It next depresses the reflex function of the cord.

3. It excites tetanus, the paralysis of coördinated reflex function of the cord progressing at the same time.

4. At last, by its influence on the cord, it produces complete paralysis.

5. It produces no effect on the motor and afferent nerves and the muscles.

Remarks and Observations on Tetanus.

The foregoing observations, coupled with those we published in the 'Lancet' for 1876 on gelseminum, suggest to us the following views regarding the true nature of tetanus. Tetanus is generally said to be due to stimulation of the cord, or the cord is said to be excited; but these expressions, we venture to think, are both erroneous and misleading.

We shall first consider strychnia tetanus. Here a slight irritation will develope rigid contraction of every muscle of the body, and this paroxysm can be excited in rapid recurrence for several days. Here no doubt an excessive discharge of nervous force does take place in the cord, and hence perhaps it may be deemed correct to say—though presently we shall have occasion to question this view—that the cord is stimulated or excited. But the effects produced by strychnia are not due to mere stimulation, for in that case the normal co-ordinated reflex movements should be retained and be more vigorously performed; this, however, does not happen, for on irritating a frog's

posterior limb, or indeed any other part, instead of inducing the usual co-ordinated movement, all the muscles of the body become rigidly contracted, and owing to the strength of the extensor muscles prevailing over that of the flexors, the hind legs, in place of being drawn up, are powerfully extended.   Here then it is evident that the impression, instead of being limited to certain parts of the cord, diffuses itself through the greater part or the whole of the cord and motor tract in the brain.   As every part of the cord is excited, all coördinated action is abolished and every muscle is contracted.   Thus in the case of strychnia tetanus, with increased nervous discharge, we have lessened resistance or greater diffusibility in the spinal cord.

We venture to suggest, and we trust our subsequent remarks will prove, that in the tetanus produced by poisons, this lessened resistance is the actual cause of the tetanus, and that there is in fact no excited condition of the cord; that, if the poison merely lessens resistance, then an irritation produces excessive evolution of force throughout the cord, and consequently strong tetanus; if the poison depresses the reflex function as well as the resistance, then an irritation induces slight evolution of nervous force throughout the cord, and consequently produces only weak tetanus; there is no excited condition of the cord.   The correctness of this view is well shown in the tetanus excited by box, gelseminum, and jaborandi.

We shall speak first and chiefly of box.   Using a concentrated solution, we find that in almost every case box excites strong tetanus, with a far greater discharge of nervous force in the cord than takes place in a normal (coördinated) reflex act; hence it is said that the cord is excited or stimulated; but the facts we are about. to adduce will, we think, show that this augmented evolution of force depends merely on lessened resistance.

After division of the cord just below the medulla through the occipito-atlantal membrane and the destruction of the medulla and brain, so as to obtain simple uncomplicated effects of box on the cord, we have shown that loss

of coördinating reflex power (cord paralysis) always preceded the tetanus by six or seven minutes. The tetanus sets in at first so slightly that only strong irritation excites it, weaker producing simply normal reflex acts ; then, as the tetanus continues and grows stronger, and becomes more easily excited, co-ordinated reflex action quickly diminishes, to be at last entirely replaced by tetanic contraction ; tetanus quickly grows weaker, and soon becomes very slight, so continuing often an hour or longer, and then ends in complete abolition of reflex action.

Thus we have diminution of reflex action *followed* by tetanus, the diminution becoming more evident as the tetanus increases, the tetanus being generally for a short time severe, and each paroxysm greatly in excess of a normal (coördinated) reflex act. Were tetanus due to stimulation of the cord, we should then have this singular and we think impossible combination, namely, first depression and then stimulation of the reflex function of the cord, this stimulation continuing concurrently with a rapid depression of co-ordinated reflex action.

Since box first weakens the reflex function before producing tetanus even when the tetanus is strong, it is probable that a larger dose is required to lessen resistance than to lessen the reflex function of the cord; hence, diminution of reflex function precedes tetanus. As soon as the quantity absorbed is sufficient to lessen resistance tetanus sets in, increasing in severity and in facility of reproduction as the resistance in the cord grows less.

In gelseminum poisoning always, and in many cases of box poisoning generally, when the extract is much diluted, tetanic convulsions are either not more marked than ordinary (coördinated) reflex acts, or are much weaker ; that
iting tetanic spasms causes a
ial cord, either no greater, and
than is expended in a co-ordi-
the amount of nervous dis-
sequent muscular contraction.
box, even when the tetanus is
weaker and weaker, and at

last they fade away to a mere quivering of the muscles. In
some cases indeed, even at its height, the tetanus is mani-
fested in little more than a quivering of the muscles. If
where the tetanus is strong it is deemed necessary to
assume that in addition to lessened resistance there must
be stimulation of the cord, we maintain that, in the in-
stances now advanced, there can be no stimulation or
heightened action of the cord, but the very opposite con-
dition, the reflex function in many cases being much
depressed. Here the stimulus evokes reflex action in a
depressed cord, but through want of resistance in the cord
the impression diffuses itself, and the reflex act itself,
though weak, is tetanic in character; and when it is borne
in mind that this weak tetanus is preceded always by some,
often by great and sometimes by almost complete de-
pression of coördinated reflex power, the foregoing view
is rendered yet more probable.

Moreover, we would urge that if tetanus is due to an
excited condition of the spinal cord, the course of the
symptoms from box poisoning should be different from
that we find it to be. In strong box tetanus, when according
to the ordinary view, the cord is stimulated and excited,
then as this condition subsides the tetanic movements
should gradually decline and at last cease, normal reflex
(coördinated) action returning; but we actually find that
the tetanic movements continue, grow weaker and weaker,
till at last all movement ceases and general paralysis
ensues. It may be said that the tetanus exhausts the
cord, and hence the convulsions grow weaker and weaker,
as the cord becomes exhausted; but this objection is
certainly without foundation; for the tetanus induced by

heightened activity of the cord, it is evident that immediately the drug begins to depress the cord tetanus should cease; for that the same substance should simultaneously stimulate and depress the same organ is inconceivable.

In these cases, where during the tetanic paroxysm the muscular contraction is not greater than occurs in a coördinated reflex act—certainly where the contraction is weaker—we must admit that the tetanus cannot depend on an excited condition of the cord, but solely on its lessened resistance, enabling an impression to diffuse itself through the greater part or the whole of the motor tract of the cord; hence all the muscles being stimulated we get tetanus.

The order in which the symptoms occur favours the view here suggested. After poisoning by box we noticed that whilst a strong irritation excited tetanus, a weak irritation produced a weakened but natural (coördinated) reflex act. The resistive power of the cord at this stage is, we submit, only slightly weakened, so that a strong impression can diffuse itself, whilst a weaker one is confined to that part of the cord naturally associated with the irritated nerve. At first, too, the tetanus is limited to the irritated limb because the resistive power being only a little weakened, the diffusion of the impression through the cord is correspondingly limited. Then as poisoning progresses, the resistive power of the cord grows weaker and weaker, and impressions diffuse more widely, till at last the the body becomes tetanised. ations to ascertain how long in after section of the cord, we l unexpected results, which we have ventured to advance. nd destroying the brain by gh the occipital foramen into llowed time for the animal to and then tested the reflex ays natural (coördinated), and

unless the blows over the back were too frequently repeated, never in any case tetanic. We afterwards tested reflex action always thrice daily, morning, noon, and at night.

Soon after coördinated reflex action began to decline, we noticed that these frogs when allowed to fall the height of an inch or two on to the glass plate became slightly tetanic. The tetanic movements were still more manifestly induced by striking the animal with the handle of the forceps over the spine between the forelegs. In the ratio of the loss of natural (coördinated) reflex action these tetanic spasms became more marked, and were more easily produced; then as coördinated reflex action further declined, so did the tetanus; but even then, after all co-ordinated reflex action had ceased, a sharp blow over the spine or on one of the extremities would elicit slight tetanic extension of the legs, with much quivering of the abdominal muscles. We made careful observations on twenty-eight frogs, and with one exception these became tetanic.

We will now describe in detail one of our ordinary cases of tetanus, taking for the purpose the first on the list. The animal was a common English frog weighing eighteen grammes. The experiment was commenced on Friday the 28th of April, the weather at the time being warm and showery.

The head of the animal having been depressed with the forefinger of the left hand, the nail readily detected the depression at the base of the skull. The point of a clean, sharp knife was then introduced so as to cut through the occipito-atlantal membrane and the subjacent medulla. The extremity of a sharpened match was then thrust into the skull through the foramen magnum so as to destroy the brain with as little loss of blood as possible. The spigot of wood was retained in position, and no hæmorrhage was visible externally. The animal suffered but slightly from the shock, and in a minute or two reflex action was found to be perfect, the whole body being drawn up when lifted by one leg. Twenty-one hours after, on striking the back in the cervical region with a pair of forceps, the legs were suddenly shot out straight, in a manner which was clearly tetanic in nature. On placing side by side with this animal a frog which had just been operated on, the contrast was very great, the legs

in the latter remaining perfectly motionless when the back was struck. By the evening of the second day (thirty hours after the operation) there was a little diminution of reflex action. At first, on striking the back no tetanus was observed, but on repeating the blow the legs were shot out as in the morning, but rather more strongly. On Sunday morning (forty-five and a half hours after the operation) a still further decline in the amount of coördinated reflex action was noticeable, although it was still fairly strong. The tetanus had increased in severity and was very readily excited. Lifting the animal ever so slightly from the table and then dropping it would at once induce a paroxysm, the legs being shot out and the web of the foot widely distended. On Monday both reflex action and tetanus gradually declined; and on Tuesday (the fifth day) had entirely ceased.

We will now describe an experiment in which the tetanus may we venture to think, be fairly denominated "very strong." There is no occasion to describe the mode of preparation, as it was identical with that already detailed. It was thought possible that the rapidity of decomposition in the warm laboratory might interfere with the development or duration of tetanus, and this animal was consequently kept during the time it was under observation in what we may call a miniature icehouse, the temperature of which ranged from 12° to 15° C. Four hours after the operation coördinated reflex action was good, and there was slight tetanus. During the remainder of this and on the following day the tetanus gradually increased in intensity, and in thirty-one hours after the operation was very powerful. The slightest touch on the back induced a violent tetanic spasm, in which the legs were forcibly extended. The paroxysm lasted more than a minute, and the animal was taken up by the legs and held out horizontally, so great was the rigidity. The individual muscles of the limbs stood out most prominently, and as the paroxysm declined could be seen rapidly alternately contracting and relaxing. This observation was at six in the evening, and at half past seven on the following morning both tetanus and coördinated reflex action had entirely ceased.

We introduce the results of our observations in the following table. In the first column we give the number of frog; in the second the date; in the third the degree of tetanus; in the fourth the time the tetanus began on merely striking the back once or twice (more frequent and more powerful irritation would always, as we have subsequently shown, depress the cord and produce tetaniform movements); in the fifth the time the tetanus

ceased; in the sixth the time it lasted; in the seventh the time normal (coördinated) reflex action ceased.

Table comparing reflex action and tetanus in frogs which had been pegged.

No. of frog.	Date.	Tetanus, degree of.	Tetanus began.	Tetanus ceased.	Tetanus lasted.	Reflex action ceased.
	1876.		Hours	Hours	Hours	Hours
I	April 28	Moderate	30	78	48	98
II	,,	Slight	20	45	25	19
III	,,	,,	30	78	48	52
IV	,,	,,	20	46	26	30
V	,,	Moderate	20	98	78	78
VI	,,	Strong	20	69	49	52
VII	April 29	Slight	10	49	39	57
VIII	May 3	Strong	7	79	72	79
IX	,,	,,	3	75	72	75
X	,,	Very strong	3	55	52	55
XI	May 6	Slight	20	75	55	95
XII	,,	,,	20	51	31	92
XIII	,,	,,	20	28	8	28
XIV	,,	Moderate	20	71	51	68
XV	,,	,,	20	71	51	68
XVI	,,	,,	3	68	65	68
XVII	May 10	Strong	28	51	23	51
XVIII	,,	Slight	48	81	33	81
XIX	,,	Very strong	28	96	68	72
XX	,,	Moderate	23	106	83	96
XXI	,,	None	...	...	...	23
XXII	,,	Very strong	4	47	43	47
XXIII	May 15	,,	4	81	77	77
XXIV	,,	Strong	4	57	53	48
XXV	,,	Slight	4	72	68	72
XXVI	,,	Moderate	4	59	55	28
XXVII	,,	Strong	4	48	44	48
XXVIII	,,	Moderate	4	52	48	48

This table shows that in four frogs the tetanus was very strong; in six strong; in eight moderate; in nine slight; and in one it was absent.

The degree of tetanus varied much, generally it consisted of the posterior legs being strongly shot out, to be immediately relaxed, but with several frogs the paroxysms when at their height, lasted from a quarter of a minute to a minute, the legs being rigidly extended, so

as to raise the animal off the table, and we could hold it out horizontally by the hind legs.

We may repeat, that directly after recovery from the shock after division of the cord, even strong irritation, unless often repeated, failed to excite tetanus, but soon after, sometimes before, normal reflex action had declined, strong irritation induced tetanus, whilst weaker irritation provoked only a coördinated reflex act. As paralysis progressed, shown in the increased weakness of the coördinated reflex acts, tetanus was more readily induced and became more marked, though even at its height the paroxysms could not be induced in rapid succession, an interval of rest being required. It will be observed that in these frogs the tetanus generally closely corresponded to the latter stages of tetanus induced by box and the whole tetanic stage of gelseminum. In some cases, however, the tetanus was strongly marked, each paroxysm lasting half a minute or even longer. These cases are comparable with the whole stage of box poisoning; for in each strong paroxysm a far greater discharge of nervous force took place in the cord than occurs in a normal (coördinated) vigorous reflex act; and therefore, according to the current views, the cord would be said to be excited. But we have pointed out that tetanus was at its height simultaneously with much weakened, coördinated reflex action, and shortly before all

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s, though less severe than
some occasions we obtained

even strong tetanus, though never approaching in severity the powerful paroxysms induced by strychnia. With the hind limbs rigidly and tonically extended the animal could be held out horizontally. After the paroxysm declined the contraction in each muscle became clonic, and thus produced jerking extension of the legs. The paroxysm sometimes continued from half a minute to a minute; the tetanus, it is true, in these cases was unusually well marked, whilst in most of our observations the attacks were much milder, though in character still clearly tetanic, and like the declining tetanus in box and the tetanus of gelseminum. If it be objected that where the paroxysms are not severe it is not true tetanus, which we get after poisoning by box, we answer, that the weaker succeed powerful and undoubted tetanic paroxysms, these gradually growing less and less, pass insensibly into a weaker and weaker form. If, then, the weaker paroxysms after box are certainly tetanic, we are clearly justified in classing as tetanic the movements just described in brainless frogs.

This tetanus, like other forms of tetanus, depends on the spinal cord. This we proved by the following experiment:—On the occurrence of tetanus we divided the sciatic nerve of one leg, and then found that whilst in the intact leg we could readily induce tetanus, we failed to excite it in the leg with the severed nerve, though some movement occurred in the thigh muscles which receive branches above the point of division of the sciatic.

It cannot be maintained, we think, that this tetanus is due to an exalted or excited condition of the spinal cord. The tetanus is preceded by loss of reflex action; it increases in strength as the coördinated reflex action grows less; it is most marked a short time before all coördinated reflex action ceases. Like the lessened coördinated reflex action it must be due, we believe, to depression of the

weaker; but with depression of reflex action we get also diminution of the resistive power of the cord, whereby impressions cease to be restricted to their proper portion of the cord, but spreading widely cause tetanic movements. At first, before much weakening of the resistance of the cord sets in, it requires a strong irritation to overcome the remaining resistance, weaker irritation still inducing coördinated reflex action. As depression of the cord progresses, the resistive power grows weaker, and the tetanic movements are more readily induced and are more · marked. As, however, the cord is much depressed they are rarely severe, and an interval is required to permit the cord to recover from the effects of the previous discharge. The depression of reflex action and resistive power progress at the same time, and consequently the total loss of power of the cord both coördinated (natural) and tetanic contraction can be induced, the coördinated by ·slight, the tetánic by stronger irritation.

The following facts also tend to show that tetanus depends on a depressed condition of the spinal cord. In these brainless frogs, at a time when a strong irritation was required to excite even slight tetanus by a repetition of the strong irritation, as for instance giving sharp blows in quick succession over the upper part of the spine, the tetanus soon became much more marked, and it was observed that, after such strong irritation, normal reflex actions were induced with more difficulty. The correct interpretation of these facts, we think, is that the shock of a succession of blows depresses the cord's resistive power whereby the irritation diffusing itself more widely causes more marked tetanic movements, and at the same time weakens normal (coördinated) reflex action. If the irritation is pushed still further, the depression becomes so great as temporarily to abolish both coördinated and tetanic reflex action, and a short time is required for the cord to recover itself.

The following observation tends to show the validity of this explanation. On striking repeatedly a living vigorous

frog strongly on the back between the shoulders, we excite at first simply coördinated reflex acts, but in a short time after each blow the posterior legs are shot out in a tetanic manner, though far less energetically than when the same phenomenon occurs in brainless frogs on the decline of reflex action. The tetaniform movement occurs only when the shock of the blows has so depressed the cord that all voluntary and normal reflex action is for the time abolished. After rest the normal (coördinated) reflex action returns, and these phenomena can be re-elicited.

It appears that some agents, like strychnia, depress only the resistive power, whilst leaving unimpaired the reflex function of the cord. Hence, with the removal of the restraining influence, slight impressions diffuse themselves throughout the cord, and produce not only a general, but likewise an excessive evolution of nervous force. Other agents depress both the resistive as well as the reflex power of the cord ; hence we get tetanus, though weak in character. In some cases the effects are developed more on the reflex function than on the resistive power, and here, as with gelseminum, we get considerable paralysis with slight tetanus. This happens also with regard to strong blows on the back, which depress both functions very considerably. Other agents depress the resistive power early and markedly ; and in a less degree the reflex function, with box for example, we get strong tetanus with slight paralysis. These views too, we think, will throw light upon the effect of slight chemical modifications of a drug on physiological action. Nerves in their constitution must differ from one another and from the cerebral nervous system, since a given poison may affect one part only of the nervous system ; for instance, the motor nerves leaving the cord unaffected. Strychnia excites powerful tetanus, and when given in very large doses simultaneously depresses the motor nerves. If converted into an ethyl compound by substituting the radicle ethyl in place of a molecule of hydrogen, strychnia no longer tetanises, but powerfully paralyses the ends of the motor nerves. We submit that

the physiological action is not entirely reversed. As strychnia it paralyses the constraining or resistive power of the cord; when converted into ethyl-strychnia, its chemical affinities being somewhat modified, it affects the motor nerves, but still it paralyses. It is not converted from a stimulant of the cord to a depressant of the motor nerves, but its chemical affinities being changed it affects the motor nerves; instead of paralysing the resistance of the cord, it paralyses the motor nerves. Its conversion into ethyl-strychnia heightens its affinities for the nerves, but lessens or destroys its affinity for that portion of the cord which restrains and localises reflex action.

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